“It’s got nothing to do with atoms”

Physical and medical evidence that the ‘nuclear’ bombings of Hiroshima and Nagasaki were faked with napalm and mustard gas

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1. Why doubt the nuclear bombing of Hiroshima?

It's got nothing to do with atoms.
Werner Heisenberg

The detonation of the a nuclear bomb above Hiroshima marks the beginning of the 'atomic age.' Isn't this an incontrovertible historic fact? Most people probably would say so. Yet, there were those who refused to believe it, at least in the beginning; and among them were leading nuclear physicists, including Werner Heisenberg [1]. In time, however, the world became convinced that the story was true. Why doubt it?

The story of the atomic bomb is certainly replete with astonishing achievements. The principle of nuclear fission was discovered only in 1938. At that time, no methods existed for isolating the fissile isotope ^{235}U,[1] which is only a minor constituent of natural uranium, but which must be almost pure for building a bomb. Even if highly enriched ^{235}U had immediately been available, one would think that first investigating its properties and behavior, then applying this new knowledge to the design of a novel bomb, and finally testing that bomb, should have taken considerable time. Indeed, some fairly preliminary experiments were going on as late as 1944. Morton Camac, a physicist who had just joined the 'Manhattan Project' fresh out of college, recounts:[2]

> *I participated in an experiment in which Uranium 235 placed in a plastic bag was dropped down the middle of a sphere with hydrocarbons. The purpose was to determine the critical setup using only the neutrons from the reaction and not from the radioactive atoms. ... The amount of*

[1] The concept of isotopes and the notation used to describe them are explained in Section A.1.

[2] The cited document [2] was obtained from a website that supports the official narrative, but I have been unable to connect it with any other of Camac's writings. Nevertheless, I tentatively judge it authentic, since it does tie in with his CV, and it is written in the jaunty yet precise style that is characteristic of reminiscing scientists. It contains some other statements that might surprise you—well worth a read.

> *Uranium was increased with each dropping. In the final dropping the neutron growth rate was so fast that the plastic melted … We were lucky that we were not killed.*

This simple procedure of trial and error differs a little from the mental picture I had formed, which featured genius theoreticians with furrowed brows, deducing the exact critical mass and the time course of the detonation from first principles alone; equipped with only chalk and blackboard, and with the largest coffeemaker the world had ever seen. Yet, only one year after this venturesome experiment, American ingenuity emerged triumphant: the first ever uranium bomb, though never once tested before,[3] went off without a hitch to obliterate Hiroshima. Does this really sound true to life, or rather like something out of Hollywood? Should we censure Heisenberg for spontaneously calling it a bluff?

Of course, this question cannot be settled by insinuations but only by the evidence, and that is what I will attempt in this book. Before going any further, however, I should point out that the book before you is not the first one to argue that the 'nuclear bomb' in Hiroshima was a hoax. A recent work entitled *Death Object: Exploding the Nuclear Weapons Hoax* [3] makes the same case, yet goes beyond it to reject the existence of nuclear weapons altogether. Its author, who uses the pseudonym 'Akio Nakatani', appears to be an expert in applied mathematics, and he claims that his own computer simulations demonstrate that nuclear bombs are impossible in principle. He does, however, not describe these calculations in detail:

> *Though I could nuke the entire orthodoxy with the scientific result … unfortunately due to archaic USA national security laws … I cannot present that openly, [thus] I am doing the next best thing, which is to compile, organize, streamline and cross-index the voluminous circumstantial evidence.*

Nakatani indeed presents ample evidence to demonstrate that the systematic fakery goes well beyond Hiroshima and Nagasaki, and I highly recommend his book. However, I will here take a somewhat different approach: instead of addressing the subject of atomic weapons in its entirety, which I am not competent to do,[4] I will focus on the scientific and medical evidence pertaining to Hiroshima and Nagasaki, which I will examine at greater depth. The findings

[3] The 'Trinity' test explosion is said to have been a plutonium bomb resembling that used at Nagasaki.

[4] I would note, however, that I consider it likely that nuclear detonations are possible in principle and have actually occurred during later bomb tests. The key problem is apparently the need to confine the critical mass long enough for the chain reaction to build. This may not be

will neither supersede nor merely duplicate Nakatani's work, but rather they will complement it.

Apart from some general works, several of which I hesitate to call 'nonfiction', the sources for this book mostly comprise scientific books and peer-reviewed articles, all of which are publicly available and have been carefully referenced. In this chapter, I will present some selected pieces of evidence; each of the topics thus introduced, and others, will be treated at greater length in later chapters.

1.1 An expert witness on the signs of destruction in Hiroshima

Alexander P. de Seversky (Figure 1.1) was a Russian-American pilot and an eminent aeronautical engineer. After the end of World War II, he was sent on an official mission to report on the results of the Allied bombing campaigns in Germany and Japan. On this tour, he also visited Hiroshima and Nagasaki. He describes his impressions from this visit in his work *Air power: key to survival* [4]. The following is quoted from the ninth chapter of this book:

> I WAS *keyed up for my first view of an atom-bombed city, prepared for the radically new sights suggested by the exciting descriptions I had read and heard. But to my utter astonishment, Hiroshima from the air looked exactly like all the other burned-out cities I had observed!*
>
> *Within an area defined by black, undestroyed houses there was the familiar pink carpet, about two miles in diameter. What is more, precisely as in Yokohama, Osaka, or Kobe, it was dotted with buildings still standing erect, with charred trees, poles, and other objects. All but one of the steel and concrete bridges were intact. A cluster of modern concrete buildings in the downtown section stood upright and seemingly undamaged.*
>
> *. . .*
>
> *I had heard about buildings instantly consumed by unprecedented heat. Yet here were buildings structurally intact, with outside and stone facings in place. What is more, I found them topped by undamaged flag poles, lightning rods, painted railings, air-raid sirens, and other fragile objects. Clearly they had weathered the blast and somehow escaped the infernal heat, as well as the alleged super-hurricane thousand-mile-an-hour wind.*
>
> *For two days I examined Hiroshima. I drove to T Bridge, which had been the aiming point for the atomic bomb. In its environs I looked for the bald spot where everything presumably had been vaporized or boiled*

practical in a weapon that must be delivered through the air, but it should be feasible using lots of concrete on the ground, or lots of rock below it.

Figure 1.1 Alexander P. de Seversky at his desk. A photograph that shows him with Harry Truman is in the background, and a copy of his book cited here [4] is in the foreground. The wikipedia page on de Seversky lists several of his books, but this one is conspicuous by its absence.

> *to dust in the twinkling of an eye. It wasn't there or anywhere else in the city. I searched for other traces of phenomena that could reasonably be tagged "unusual." I couldn't find them.*

In his subsequent chapter, entitled *Atomic hysteria and common sense*, de Seversky writes about the reactions to his report from Hiroshima in the United States:

> THE STORY *sketched in the preceding chapter obviously was different from the one then being told virtually in unison by press, radio, and scientists. Against the prevailing hyperbole it must have sounded more incredible than I suspected. But it was the only story I could conscientiously tell when I was questioned by newspapermen in Tokyo and back home in America.*
>
> *I did not "underrate" the atom bomb or dispute its future potential. Certainly I did not dismiss lightly the infernal horror visited on Hiroshima and Nagasaki. As an engineer, I limited myself to an analysis of the demolition accomplished by particular bombs exploded in a particular way. These one-man observations I embodied in a formal report to the Secretary of War, who released it to the public. In addition I wrote several articles on the subject.*

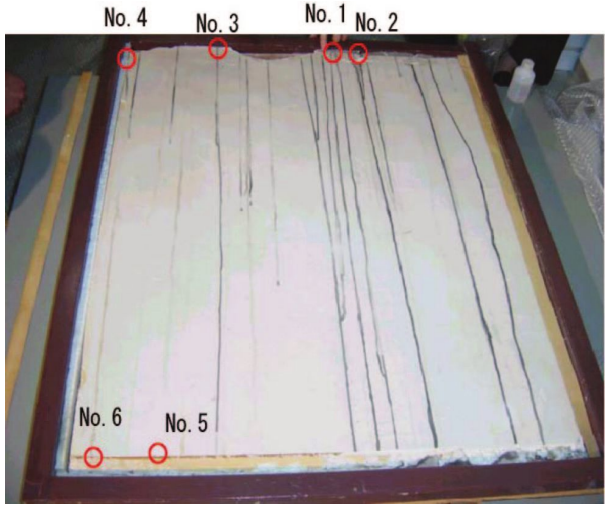


Figure 1.2 Plaster board contaminated with black rain streaks (photograph taken from [5]). Circles indicate locations that were sampled. Sample 3—the sample that yielded the highest amounts of the telltale isotopes (see text)—is located not on the face of the board but rather on its upper edge.

> *Whereupon all hell broke loose over my sinful head. My findings were pounced upon by all sorts of people in angry fury, on the air, in the press, at public forums; scientists who hadn't been within five thousand miles of the atomized cities solemnly issued condemnations of my heretical views. Almost for the first time in my career I found myself in the position of a "conservative" under fire from "extremists."*

As is clear from de Seversky's protestations, he did not question the reality of the atomic bombs. His only 'sin' was to faithfully report the lack of evidence of their distinct and apocalyptic effects; the bombed cities of Hiroshima and Nagasaki had impressed him in much the same way as the many cities destroyed by conventional air bombing which he had visited before.

We will return to the question of what visible traces a nuclear blast should or should not have left behind in later chapters; here, we will simply note that the visible signs of Hiroshima's destruction were compatible with a conventional bombing raid. Let us now sample some proper, quantifiable physical evidence.

1.2 The missing uranium

The Hiroshima bomb ('Little Boy') purportedly contained some 50 kg of uranium, with the fissile ^{235}U isotope enriched to about 80%. Furthermore, of those 50 kg, less than 1 kg is said to actually have fissioned. Where did the other 49 kg go?

Several scientific studies have looked for this uranium, but all have come up short. One such study was carried out by Shizuma et al. [5]. The authors obtained samples from an interior plaster board of a house whose roof had been blown off in the attack, and which had been soiled by the notorious 'black rain' that came down a short while after the bombing. The plaster board in question is shown in Figure 1.2.

The traces left by the black rain were analyzed for uranium using mass spectrometry, which separates chemical elements and their isotopes according to atomic weight. Because uranium has significant abundance in nature,[5] the question arises how much, if any, of the uranium detected in the samples might be due to natural background, and how much is derived from the bomb. Since natural uranium contains > 99% ^{238}U, while bomb uranium should be 80% ^{235}U, this question can readily be answered: the higher the isotope ratio $^{235}\mathrm{U}/^{238}\mathrm{U}$ in the sample, the greater the fraction of bomb uranium.

What is the answer?

In most of the samples studied, the isotope ratio deviated only very slightly from the natural one, indicating negligible amounts of bomb-derived uranium. The highest ratio was observed with a sample taken from the upper edge of the plaster board, which unlike the face of the board had not been wiped down by the house's residents. The ratio observed in this sample—0.88%, vs. 0.72% in natural uranium—indicates that, of the total uranium in the sample, *just 0.2% would be derived from the bomb.*

This value surely is surprisingly low; so low, in fact, that one might wonder if these samples contained any bomb-derived uranium at all. Could it be that those black stripes were not what they were believed to be—that they had no relation to the black rain at all? Two arguments can be raised against this. Firstly, mass spectrometry is highly accurate—a deviation in the uranium isotope ratio as high as observed would not arise through a statistical fluke.

Secondly, in addition to ^{235}U, the authors also detected small amounts of radioactive cesium (^{137}Cs) in those same samples. This isotope is one of the main products of nuclear fission. Its radioactive half-life is much shorter than those of ^{235}U and ^{238}U—only 30 years. This is far too short for it to occur in nature; therefore, ^{137}Cs is a telltale sign of artificial, man-made nuclear fission.

Should neither of the above arguments satisfy you, be advised that the number reported by Shizuma et al. [5]—bomb-derived uranium amounting to just 0.2% of the natural background—is the *highest* figure reported in any of the

[5] Since the natural abundance of ^{235}U in uranium ore is only about 0.72%—with most of the rest being ^{238}U—preparing that amount is no mean feat in itself. In Section 2.6, I will argue that the technology most likely did not exist at the time; however, for now this question will be set aside.

studies on Hiroshima fallout that I could find. Thus, if we reject this number as invalid for being too low, we must reject all those other studies also, and we are left without any evidence at all of ^{235}U in the fallout.

We can conclude that both ^{235}U and ^{137}Cs fell upon Hiroshima on August 6, 1945. The very low abundance of ^{235}U in the fallout, however, fits very poorly with the story of the purported nuclear blast, and indeed this notion will be laid to rest altogether by a more in-depth analysis of published scientific data in later chapters. For now, let us turn to some witness testimony about the event itself. Surely, those dramatic accounts of an unprecedentedly violent explosion will tell the story, and obviate the need to puzzle over dirt on plaster boards?

1.3 Eyewitness accounts of the attack

Eye witnesses of the bomb are unanimous that the atomic bomb produced an intense, blinding flash, quickly followed by an enormous bang. Or are they? Here are some quotes from John Hersey's famous book, *Hiroshima* [6]:

> *Then a tremendous flash of light cut across the sky. Mr. Tanimoto has a distinct recollection that it traveled from east to west, from the city towards the hills. It seemed a sheet of sun. . . . He felt a sudden pressure, and then splinters and pieces of board and fragments of tile fell on him. He heard no roar. (Almost no one in Hiroshima recalls hearing any noise of the bomb. But a fisherman . . . saw the flash and heard a tremendous explosion; he was nearly twenty miles from Hiroshima . . .)*

Whether nuclear or not, it is astonishing that an explosion should be audible from twenty miles away, but inaudible from almost directly underneath it. Could it be that all those close to the detonation simply had their ears shattered before they even could perceive the sound? Apparently not—Ishikawa et al. [7, p. 126] state that only 1% of all hospitalized patients in Hiroshima had ruptured eardrums (but 8% of those in Nagasaki; both values are within the range observed in conventional bombings [8]).

Another interesting source is Keller [9], an American physician who was working in Japan during the fall of 1945. He writes:

> *The information presented in this report was obtained from studies on 21 patients who were admitted to the Osaka University Hospital in late August and early September 1945 suffering from an alarming malady designated atomic bomb disease by the Japanese. I observed, examined and followed approximately half of the patients, while information on the remaining patients was taken from the hospital records.*

> *Only 5 patients recalled experiencing a definite concussion wave at the time of the atomic bomb explosion. One of the 5 who was in a wooden building about 50 meters from the center of the explosion was thrown 12 feet by the blast as the building collapsed. The 2 victims who were outdoors had contrasting experience in that 1 was knocked unconscious while the other 1 felt no blast.*
>
> *Three patients recall hearing a noise "like the sound of an explosion." One described a noise that sounded "like a falling bomb," and 2 said the noise they heard at the time of the atomic bomb explosion was a sound "like rain." Two stated that they heard no definite sound of an explosion, while the remaining 13 were uncertain.*
>
> *Nine patients were conscious of a "flash of light" when the bomb exploded. One of the 9 described the light as being green. Three of the remaining 12 patients experienced no sensation of light, while the other 9 case records do not specify one way or the other.*

There is no need to belabor the stark contradictions in this testimony, but I do want to draw your attention to the first of Keller's patients—the one who was just 50 meters from the hypocenter, shielded from radiation by nothing more than a wooden house. If there had indeed been a proper nuclear detonation, he should have been killed immediately, or at least very rapidly, by the blast, the heat, and the radiation; but here he is, some four weeks later: hospitalized and 'alarmingly' ill, but alive enough to tell the tale.[6]

The remainder of Keller's account consists of clinical and laboratory findings on what he interprets as radiation sickness. When examined in detail, such observations also fail to support crucial aspects of the official story, as will be shown in Chapter 5. For now, we note that the available witness testimony on the blast and the flash expected of a proper nuclear detonation is inconsistent.

One aspect that we have not yet considered is the 'mushroom cloud' that rose above Hiroshima after the attack. Without going into detail, I will just note that such clouds—referred to as *flammagenitus* or *pyrocumulus* clouds—are not limited to nuclear detonations, but are also seen above wildfires or burning cities. Readers interested in this subject may consult Nakatani [3], who gives a good general discussion and also dissects the many inconsistencies in the reports on the mushroom clouds observed over both Hiroshima and Nagasaki.[7]

[6] While one might dismiss a single such case report as spurious, Section 5.4.1 will show that there are more.

[7] In fact, even the New York Times has reported—on May 24th 2016, under the heading *The Hiroshima Mushroom Cloud That Wasn't*—that the mushroom cloud was caused by the burning of the city rather than the nuclear detonation.

1.4 What really happened on that day?

If we maintain that no actual nuclear blast occurred at Hiroshima, we must provide an alternate explanation for the destruction, the radioactive fallout (small as it may be), and also for the medical findings in numerous victims that broadly resemble those of exposure to intense irradiation.

1.4.1 Destruction of the cities with incendiary bombs. These questions are also discussed by Nakatani [3], who proposes that the city was destroyed by a conventional bombing raid. Most buildings in Japanese cities were constructed from wood. Accordingly, in their conventional bombing raids, the Americans relied mostly on incendiaries, which according to the U.S. Strategic bombing survey [10] included both 'oil-gel' (napalm) and thermite. As we will see later, the evidence suggests that both types were used in Hiroshima and Nagasaki also; and even though scattered, some witness reports of incendiary bombs falling on Hiroshima and Nagasaki can be found. Nakatani also discusses a non-nuclear pyrotechnical scenario for the 'flash', which, even though not perceived by all witnesses, does seem to figure more commonly in victim testimony than the 'bang'.

1.4.2 Dispersal of reactor waste to create some fallout. Finally, Nakatani posits that some radioactivity—probably reactor waste—was dispersed using conventional explosives, relating that such a device—known as a 'dirty bomb'—had previously been tested in New Mexico. Chapter 2 will show that a dirty bomb indeed fits the published scientific findings on 'Little Boy's' radioactive fallout much better than does the official story of the nuclear detonation.

1.4.3 Use of mustard gas to fake 'radiation sickness'. Keller [9] reports that many Hiroshima victims suffered from bone marrow suppression and other symptoms that are commonly observed in patients exposed to strong irradiation, be it by accident or for treatment; and these statements are confirmed by many other medical case studies and surveys. The very low amount of dispersed radioactive material apparent from studies such as Shizuma et al. [5] cannot account for these observations.

Nakatani recognizes this incongruity and proposes that clinical reports of radiation sickness are mostly fabricated, although he suggests that a dirty bomb might have produced some real cases. I concur in principle that much of the science that surrounds this event is fraudulent, and I will discuss some specific examples in later chapters. However, the medical reports are too numerous and come from too many independent sources to be so nonchalantly dismissed, and in fact they can be readily explained by the use of poisonous gas. This possibility

was raised early on by Dr. Masao Tsuzuki, the leading Japanese member on the U.S.-Japanese 'Joint Commission' of medical scientists convened to investigate the aftermath of the bombing. Sey Nishimura [11] quotes from a 1945 article by Tsuzuki:

> *... immediately after the explosion of the atomic bomb, some gas permeated, which appeared like white smoke with stimulating odor. Many reported that when inhaled, it caused acute sore throat or suffocating pain.*

According to Nishimura, Tsuzuki's position concerning the gas attracted the attention of the U.S. military censors, who for the sake of 'public tranquillity' struck out the following passage from his manuscript:

> *Considering from various points, generation of something like poisonous gas accompanying the explosion operation is conceivable, and it is not hard to conjecture that there were perhaps war victims who died of these poisons. At present we have no clue whether it was devised on purpose so as to radiate something like poisonous gas. If I have a chance, I'd like to put a question to America on this matter.*

Again according to Nishimura, Tsuzuki nevertheless reaffirmed his position in another report six years later:

> *... everyone experienced inhalation of a certain indescribable malodorous gas. This may be considered city stench, which was induced by fierce wind from the explosion; a part of it might have originated from electrolytes generated by application of radioactivity to air. What this so-called "gas" is, is not clear. But it is not unthinkable that it could be invasive to the human body.*

Tsuzuki's conjecture on the radiogenic origin of the gas is sound in principle: intense radiation traveling through air can indeed produce pungent, aggressive gases such as ozone and oxides of nitrogen. However, assuming that no nuclear detonation actually happened, we can rule out this possibility, which means that any poisonous gas present must have been dropped in finished form during the air raid. It is interesting to note that the first independent journalist to report from Hiroshima, the Australian Wilfred Burchett [12],[8] also brings up poison gas:

[8] This report first appeared under the name 'Peter Burchett' in the *Daily Express* on September 5th, 1945.

My nose detected a peculiar odour unlike anything I have ever smelled before. It is something like sulphur, but not quite. I could smell it when I passed a fire that was still smouldering, or at a spot where they were still recovering bodies from the wreckage. But I could also smell it where everything was still deserted.

The gas plagued the people even four weeks after the event:

And so the people of Hiroshima today are walking through the forlorn desolation of their once proud city with gauze masks over their mouths and noses.

The Japanese interviewed by Burchett conflated it with radioactivity:

They believe it [the smell] is given off by the poisonous gas still issuing from the earth soaked with radioactivity released by the split uranium atom.

Their conjecture on the origin of the gas must be false, for there is no plausible mechanism by which radiation or fallout from a nuclear bomb could produce this sort of lingering fumes.[9] However, this should not mislead us into discounting their perceptions altogether; surely no one toiling in hot summer weather will wear a face mask without reason. What kind of gas would fit this entire scenario?

The most likely candidate is sulfur mustard, which had been used as a chemical weapon in World War I, and which was so used again more recently by Iraq in its war against Iran. Sulfur mustard mimics both the acute and the chronic effects of radiation on the human body. In particular, like radiation, mustard gas damages the bone marrow, the hair follicles, and other rapidly proliferating tissues; and this commonality was already well understood at the time [13].[10]

An oily fluid, sulfur mustard can evaporate slowly over time; its smell resembles that of 'garlic, addled eggs, or oil-roasted vegetables' [15] and is also sometimes described as sulfuric. It can persist in the environment for considerable periods of time [16], which would explain that Burchett still noted its stench and its effects when he visited Hiroshima in early September.

[9] As stated above, some ozone and nitrogen oxides might well be produced in a nuclear blast, but they would be short-lived.

[10] Substances with such properties are sometimes referred to as *radiomimetic* [14]; and the cytotoxic effects of both radiation and radiomimetic chemicals are exploited in the treatment of cancers and leukemias.

1.4.4 Preparedness of the U.S. military for the use of mustard gas. The U.S. had stockpiled sulfur mustard in World War II and had even conducted experiments on some of their own soldiers.[11]

In 1943, a large number of U.S. servicemen and civilians had been killed by the poison when it was released from containers carried on an American military transport ship during a German air attack, in the Italian port city of Bari.[12] This disaster would have been fresh on the minds of the military brass when the plans for the fake nuclear bombings were first sketched out.[13]

While the effects of mustard gas resemble those of radiation in several ways, there nonetheless are differences between the two. A nuclear detonation will produce radiation predominantly in the form of γ-rays and of neutrons, both of which are highly penetrating and thus have marked effects on rapidly proliferating tissues inside the body; they will destroy the bone marrow at dosages well below those that will severely harm the skin, the lungs, and even the intestines (though these are second in susceptibility only to the bone marrow). Mustard gas, in contrast, must be taken up through the skin or the mucous membranes of the lungs or intestines, and in the process it will produce marked and early symptoms of damage to these organs. You may have read accounts like the following, again taken from John Hersey [6]:

> *The eyebrows of some were burned off and skin hung from their faces and hands. … He reached down and took a woman by the hands, but her skin slipped off in huge, glove-like pieces.*

While standard lore explains such lesions as thermal 'flash burns' caused by the light radiating from the bomb, they really do not fit that description. Instead, they are strikingly similar to those described by the military physician Alexander [18] in the mustard gas victims at Bari:

> *In many cases large areas of the superficial layers of the epidermis were separated from their deeper layers and torn loose … The pathologists re-*

[11] According to the book *Veterans at Risk: The Health Effects of Mustard Gas and Lewisite* [17], this program involved more than 60,000 military personnel; in a later survey of these subjects, only 12 out of 257 respondents reported no adverse health effects.

[12] Alexander, the medical officer who oversaw the treatment of the mustard victims at Bari, writes that 83 servicemen died of the poison in hospitals [18], but also indicates that the overall death toll was likely higher (e.g., he states that all those aboard the ship that had carried the sulfur mustard were killed). The civilian death toll was likely much higher [19, 20].

[13] Interestingly, according to Brodie [21], research on reactor development, military use of fission products, and mustard gas toxicity were all concentrated at the University of Chicago in the early 1940s. In some of these studies, the effects of mustard gas and of nuclear fission products on lung tissue were compared side-by-side in animal experiments [22].

peatedly noted that these layers of the skin were dislodged upon handling of the body ... As the superficial skin layers were stripped loose they often took their surface hair with them.

Similar descriptions were given by other physicians [23, 24]. The characteristic skin lesions are but one sign that distinguishes mustard gas poisoning from true radiation sickness; there are others, which may be less graphic yet are more specific and decisive. As we will see later, clinical and pathological reports from Hiroshima contain a wealth of evidence that clearly points to sulfur mustard or a closely similar poisonous gas, rather than radiation, as the cause of 'radiation sickness' among the victims in Hiroshima.

Alexander further notes:

Thermal burns were readily distinguished from the chemical burns. There were a small number of cases that sustained minor thermal burns in addition to their mustard injuries.

Thermal burns must have occurred in those victims whose wooden houses had been set afire and collapsed around them. In addition, however, it is likely that many of the burns in Hiroshima and Nagasaki were inflicted by napalm or a very similar incendiary; this will be discussed in more detail in later chapters.

In summary, therefore, the thesis of this book as to what happened in Hiroshima is the same as that of Nakatani [3], but augmented with sulfur mustard, which was used to mimic in the victims the symptoms of exposure to strong radiation.

1.5 The evidence in the case

While the physical and medical evidence will be more fully presented in later chapters, it is useful to consider beforehand how different kinds of findings relate to the overall case.

1.5.1 Evidence that directly disproves the nuclear detonation. Some findings prove that physical and medical effects expected of the purported nuclear detonation did not in fact occur. Among the examples introduced above, we can cite the lack of ^{235}U in the fallout (Section 1.2), the absence of characteristic signs of destruction in the city (Section 1.1), and the survival of people who were practically right at the hypocenter, protected from the blast and the radiation by nothing more than a Japanese style wooden house (Section 1.3).

Another important finding in this category is the absence of retinal lesions in survivors who reported having looked directly at the flash. As we will see later, there are both case reports and experimental studies to show that these

survivors should all have had their retinas severely burned and scarred, had they indeed looked at a real nuclear detonation.

1.5.2 Evidence that cannot be accounted for by the atomic bomb. The official story of Hiroshima states that the city was destroyed by a single atomic bomb and nothing else. Thus, any kind of destruction or trauma that is *not* explained by this single bomb also contradicts the official story, even though it does not disprove the detonation of an atomic bomb outright.

A crucial finding in this category is the occurrence of 'radiation sickness' among those who were not close to the alleged bomb detonation. All orthodox sources on the effects of the Hiroshima bomb—see for example Okajima et al. [25]—agree that levels of radiation sufficient to induce acute radiation sickness occurred only during the detonation itself, and only within at most 2,000 m of the hypocenter;[14] in contrast, the residual radioactivity due to fallout and neutron capture remained below this threshold both at the hypocenter and in the Koi area of the city, which is some 2 km from the hypocenter yet received the highest levels of fallout. Nevertheless, numerous cases of 'radiation sickness' have been reported in people who were more than 2,000 m away from the 'blast' or even outside the city altogether. The victims within this group often fell sick after participating in rescue and recovery efforts in the inner city in the aftermath of the bombing. Two such cases, both with deadly outcome, are described in an early report by the International Red Cross [27]. Larger statistics that amply support this contention can be found in reports by Oughterson et al. [28] and Sutou [29].

A piece of physical evidence in this category is the reddish residue in the burnt-down area of the city. De Seversky notes this 'pink carpet' as a common occurrence in conventionally firebombed cities and ascribes it to construction steel oxidized and dispersed in the fire [4]. However, while iron may deform in a regular fire, it will not readily melt and disperse. Moreover, most buildings in Hiroshima were of wooden construction and most likely contained only small amounts of iron to begin with.

A more plausible cause of the pink carpet is *thermite*, a pink-colored powder which combines metallic aluminum with iron oxide and is used as an incendiary.[15] That the U.S. were using thermite at this stage of the war is clear from

[14] The minimum dose to induce acute radiation sickness is approximately 1 Sv [26]. Lower doses might cause long-term effects such as increased incidence of leukemia and cancer, but this does not matter in the current context.

[15] The exothermic reaction that occurs in this mixture is $2\,\mathrm{Al} + \mathrm{Fe_2O_3} \longrightarrow \mathrm{Al_2O_3} + 2\,\mathrm{Fe}$. The combustion does not produce any gas that would expand and disperse the released heat, which thus remains concentrated in a confined spot. Thermite can be used to weld or melt steel, and

the U.S. Strategic Bombing Survey [10], which reports that some 1,500 tons of thermite-filled bombs were dropped on the fairly small city of Hachioji only five days before the Hiroshima bombing. Unreacted residue from such an enormous quantity would readily explain the pink carpet noted by de Seversky [4] and also by other sources [27, 31].

1.5.3 Evidence of the use of mustard gas. This category is a special case of the previous one, but it is important enough to be highlighted separately. In addition to the skin forming blisters and being torn loose (Section 1.4), there is abundant evidence of immediate, acute affliction of the airways and the intestines, which in the course of acute radiation sickness should be affected only at a later stage or not at all. The involvement of these organs is clear both from clinical descriptions and from autopsies of bombing victims.

Importantly, mustard gas also mimics the typical manifestations of radiation sickness such as bone marrow suppression and epilation, and it can persist in the environment for weeks or even months [13, 32]. Thus, mustard gas accounts for 'radiation sickness' not only in those who were in the city at the time of the bombing, but also in those who entered it in the aftermath. Moreover, in can account for some atypical symptoms which do not fit the textbook pattern of true radiation sickness; it explains the entire picture and succeeds where nuclear radiation falls short.

1.5.4 Fabricated evidence of the nuclear detonation. The case *for* the nuclear bomb is, of course, supported by an endless stream of government-sponsored scientific studies. For example, there are dozens of reports on the formation of ^{60}Co and other radioactive isotopes near the hypocenter, which is ascribed to the neutron radiation from the nuclear detonation. Similarly, thermoluminescence in samples of ceramic materials is adduced as proof of the γ-irradiation released by the detonation.

Taken at face value, such studies indeed prove that a large amount of both γ-rays and neutrons was released at Hiroshima, which clearly supports the story of the nuclear detonation and flatly contradicts the negative evidence discussed above. We are thus forced to choose sides. On what basis can we make this choice?

If we assume that no blast occurred, then we must conclude that the evidence of neutron and γ-radiation is fabricated. This is not technically difficult; in fact, the studies in question commonly employ control and calibration samples that were generated by exposing inactive precursor materials to defined doses of

a particularly effective preparation of thermite was used in the destruction of the World Trade Center on September 11th 2001 [30].

laboratory-generated neutron and γ-radiation. The only difficulty is a *moral* one—we must accuse either the scientists themselves or a third party such as a government or its secret service of substituting artificial samples for the real ones. In this context, it is noteworthy that none of the studies I have seen documents the chain of custody of its samples; it is not clear who had access to the samples at which times.

If, on the other hand, we assume that a nuclear blast *did* occur, and furthermore that *only* this blast occurred, then we have to conclude that some people inexplicably survived deadly doses of radiation, whereas others succumbed to acute radiation sickness without significant exposure. A third miracle is needed to explain that all people who looked at the flash of the detonation escaped with their retinas unhurt.[16]

Between moral embarrassment and scientific impossibility, the only rational choice is the former. We all expect the strength of character to make such choices correctly of those whom we entrust with the adjudication of criminal cases; in a case like this, we are all members of the jury.

1.5.5 Missing evidence. Evidence that has been lost or was not collected in the first place cannot, of course, directly support either side of an argument. It will matter only on a meta-plane, and only to those who would entertain the possibility of its deliberate suppression; readers familiar with the controversies surrounding the Kennedy murders or the twin tower collapses will likely recognize the theme. While in my own view the missing evidence rounds out the case, it is not a logically essential element.

Some choice examples of disappearing evidence are provided by the physicist John A. Auxier [33]. While he remarks that "it is difficult to realize the passion that prevailed after the war for secrecy about all information concerning nuclear bombs," he nevertheless accepts at face value the official story that had to be nurtured by such secrecy, and he dedicated a large part of his own career to the arduous work of filling the gaps in the accepted picture of the radiation doses released and received at Hiroshima and Nagasaki.

Considering the great novelty of the atomic bombs, the U.S. military would certainly have been highly interested in measuring exactly the force of their detonations. To this end, the planes dropping these bombs were accompanied by others that dropped instruments for recording the shock waves of the explosions. Since the strength of the shock wave decreases with distance, it

[16] There are reports of transient loss of vision, which are entirely consistent with the known effect of mustard gas on the cornea of the eye. In contrast, retinal damage should have been irreversible.

was important to know precisely the distance between the bombs and these instruments. In this context, Auxier notes:

> *If there are need, interest, and credentials, information about bombing missions in World War II can be obtained in great detail from Air Force records. For a given mission, the aircraft identification numbers, names of crew members, types of bombs, bombing altitude, winds aloft, approach direction, and indicated and true airspeed can be found. There are, however, at least two exceptions to this ... The records for the two most important bombing missions in history are incomplete and inaccurate to a degree beyond comprehension.*

In addition to the strength of the explosion, the intensity of the radiation produced should also have been of great interest. It is therefore peculiar that radiation measurements in Hiroshima by American teams began only in October, at a time when most of the radioactivity left behind by the bomb would already have vanished. However, several Japanese teams performed measurements on their own initiative. Among them was a group from Kyoto University that included the physicist Sakae Shimizu, who carried out some very early measurements pertaining to the dose of very high energy neutrons [34]. How did the Americans treat this valuable evidence? Says Auxier:

> *Unfortunately, soon after the war ended and while Dr. Shimizu's studies were still underway, the U.S. occupation force confiscated the cyclotron and all apparatus and records that laymen would consider to be related to atomic bomb research. Included in the latter were the radium source [required for calibrating instruments for measuring radiation] and all the notebooks of data. Through the handwritten receipt that had been given Dr. Shimizu, the confiscating officer was identified some 12 years later, and, by the cooperation by the Army records staff, he was located in civilian life. However, soon after receiving the materials from Dr. Shimizu, the officer was ordered back to the United States with little time for an orderly changeover. He turned everything over to a lieutenant colonel or major whose name he could not recall. Further research through Army records has failed to identify this man or to locate any trace of the notebooks or radium source. It is likely that they are in some file in one of the many record depositories and that some day they will be found. In the meantime, the job that would have been much easier with these records went on without them.*

Surely an astonishing imbroglio of mishaps and incompetence. It should be added that the Kyoto cyclotron was not merely 'confiscated' but physically destroyed, as was every other cyclotron in the country [35, 36]. This draconian measure of course severely crippled the Japanese scientists' ability to carry out any sort of in-depth study on the physical effects of the atomic bombs.[17] At the same time, their investigations into the medical effects were hamstrung by the confiscation of all tissue and organ samples that had been collected from bombing victims by Japanese pathologists [37]. These materials were returned to Japan only several decades later; and while in American custody, they only made a single appearance, limited and belated, in the scientific literature [38].

The examples in this section may suffice to outline a map on which to place the various kinds of evidence in the case. In the subsequent chapters, we will explore this evidence at greater depth.

[17] According to the Japanese nuclear physicist Nishina [36], the American Secretary of War Patterson blamed the destruction of the cyclotrons on the 'mistake' of some nameless Pentagon underling. I have found no record of anyone being held accountable.

2. The nuclear fallout of the Hiroshima and Nagasaki bombings

While the atomic bombs in Japan had unprecedented explosive force, they were dwarfed by the much more powerful ones that were developed and tested in subsequent decades. According to Carter [39], the nuclear bomb tests during the 1950s, 60s, and 70s totaled 905 in number and 344 megatons in yield. Collectively, these tests produced a large amount of radioactive fallout, much of which was dispersed all over the Northern hemisphere, and which can be ubiquitously detected with modern, sensitive instruments.

If we want to determine how much fallout remains at Hiroshima and Nagasaki from the original bombs, we must take the global fallout into consideration. There are two ways of doing so. Firstly, we can look for samples that were secured, or at least protected, early on, before they could become contaminated with the global fallout. Secondly, we can exploit the distinct nature of the purported Hiroshima bomb, which used highly enriched ^{235}U as its fuel, while all later bombs and bomb tests used plutonium (^{239}Pu) instead.[1]

The fission products which form from ^{235}U and ^{239}Pu are quite similar; in particular, the widely used fallout tracer ^{137}Cs is found with both. However, unfissioned ^{235}U itself, when exceeding the natural isotope ratio relative to ^{238}U, would be a specific tracer for the Hiroshima bomb. The study by Shizuma et al. [5] cited earlier applied both of these principles: it quantified ^{235}U in samples touched only by local but not by global fallout. This circumstance earned it preferred treatment.

2.1 Uranium isotopes in soil samples

Apart from ^{235}U and ^{238}U, several other uranium isotopes exist that have low abundance, yet can be of value in understanding what did or did not happen at Hiroshima. Sakaguchi et al. [42] examined the abundance of ^{236}U, which forms from ^{235}U by neutron capture without fission. A complicating factor, however,

[1] Non-enriched uranium can be used as a component of hydrogen bombs and has been detected in fallout shortly after such bombs were tested [40], but this will not cause upward deviations of the ^{235}U/^{238}U isotope ratio.

Figure 2.1 Area affected by black rain in and near Hiroshima. The areas of heavy and light black rainfall extend in NWN direction from the hypocenter (red dot) and are indicated by solid and dashed red outlines, respectively. Concentric rings indicate distances of 10, 20, and 30 km from the hypocenter. Drawn after a similar map in [41]. The studies cited in this chapter mostly used soil samples from within the heavy black rain area.

is that ^{236}U also arises through radioactive decay of ^{240}Pu, the second most abundant plutonium isotope. Since ^{236}U decays very slowly, the method used in this study was mass spectrometry.

Starting from conventional estimates of bomb size, degree of ^{235}U enrichment, and fission yield, the authors estimate that 69 g ^{236}U should have been generated in the detonation, and they set out to look for it in the area affected by the black rain. At this point, you might not be surprised to learn that they do not find it; or more accurately, they do find some ^{236}U, but after comparison with plutonium levels and with samples from a control area in Japan taken to be unaffected by 'Little Boy', they conclude that all of it must be attributed to the global fallout. To explain the lack of a discernible local contribution, they assume that the black rain transported only a very small fraction of the radioactive matter generated in the blast.[2]

[2]The authors also found total fallout in the control area to be about twice *higher* than in Hiroshima. Readers with common sense surely will understand that this tells the story and skip the rest of this chapter; readers without it must persevere.

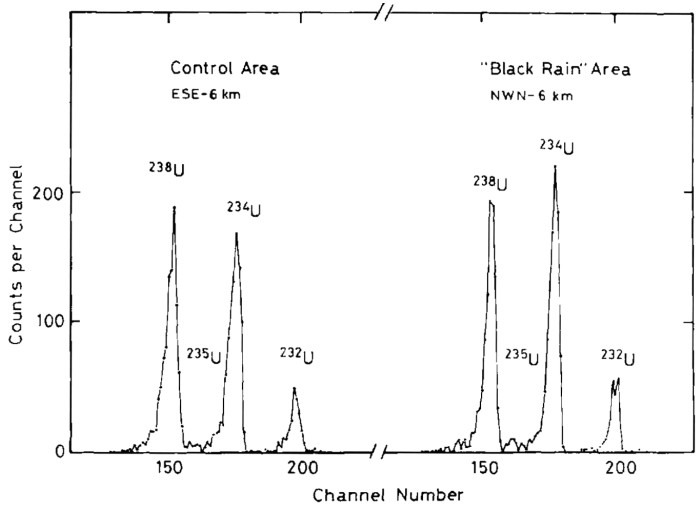


Figure 2.2 α-Ray spectra of uranium extracted from soil samples using 0.1 N nitric acid (taken from Takada et al. [41]). The α-particles emitted by the various uranium isotopes are distinguished by their characteristic energies, which correspond to 'channels' along the x-axis; the abundance of each isotope is represented by the area under its peak (rather than the peak height). See text for details.

The major component of natural uranium, ^{238}U, undergoes α-decay, which is followed rapidly by two β-decays; this yields ^{234}U. The half-life of ^{238}U is very long (4.47 billion years), whereas that of ^{234}U is comparatively short (246,000 years). At steady state, ^{234}U will decay exactly as fast as it is formed through decay of ^{238}U. Therefore, if we stick a sample of natural uranium into a radiation counter, we should measure equal activities for both these isotopes. The relation should be different, however, with enriched uranium, as was supposedly used in the Hiroshima bomb. Because ^{234}U is close to ^{235}U in atomic weight, both isotopes should have been enriched together relative to ^{238}U. Assuming that in the Hiroshima bomb ^{234}U, like ^{235}U, was enriched by a factor of about 100 over its natural level, whereas ^{238}U was reduced by a factor of 5, the activity (but not the abundance) of ^{234}U in the bomb material should exceed that of ^{238}U by some 500 times. Therefore, the activity ratio of ^{234}U/^{238}U should be a sensitive probe for the detection of residual bomb uranium.

A very careful study that employed this probe was carried out by Takada et al. [41]. The samples consisted again of soil from the black rain area. What makes this study particularly interesting is the attempt to separate bomb-derived uranium from that which constitutes the natural background. The bomb fallout should only adhere to the surface of the soil particles, whereas the natural

uranium should mostly reside within them. Thus, to extract the fallout, the soil samples were gently leached with dilute acid, which should strip only a shallow, superficial layer from the particles; the background was then recovered by dissolving the residue with concentrated acid.

In the fraction recovered with dilute acid, ^{234}U activity indeed exceeded that of ^{238}U—but only by a factor of approximately 1.15; compare this to the factor of about 500 expected for pure, highly enriched bomb uranium. This slight excess was observed only with samples from the black rain area, but not with those from a control area outside it.[3] The activity of ^{235}U, which in pure bomb-uranium should exceed that of ^{238}U some 30 times, remained very low in all samples (see Figure 2.2).

As with the study by Shizuma et al. [5] cited before (Section 1.2), we have evidence of a small yet distinct deviation from the natural uranium isotope distribution; and the magnitude is similar between both studies. There are two explanations in principle—namely, either that a minuscule amount of highly enriched bomb uranium was diluted to near nothingness by natural background, or that the degree of ^{235}U enrichment in the dispersed artificial material was much lower than announced. Takada's failure to detect a higher degree of enrichment even when taking steps to concentrate the bomb uranium clearly militates in favor of the second alternative.

Considering this evidence, as well as the state of technology as it then prevailed (see Section 2.6 below), I feel certain that no highly enriched ^{235}U was released at Hiroshima. However, here is how to prove me wrong: obtain a sample of pristine glacier ice, and analyze it for ^{235}U and ^{238}U. This has been done for both cesium and plutonium on a sample from Ellesmere Island in the Canadian arctic, and it is claimed that the imprint of the Nagasaki bomb is detectable in the layer of ice that was deposited in the year 1945 [43]. Such a sample should be largely free from terrestrial background, and using the exquisite sensitivity of modern mass spectrometry, the isotopic signature of 'Little Boy' should be unmistakable.[4]

2.2 Cesium and uranium in samples collected shortly after the bombing

Since global fallout is rich in plutonium and in radioactive fission products such as ^{137}Cs, soil samples that were protected from it should have great value for

[3] As discussed by Takada et al. [41], determination of the true ratio is complicated by the slight variation of the two isotopes' abundance in different types of soil, which is caused by a slight difference in solubility. However, in the current context, this variation is inconsequential.

[4] Some small amount of dust will be present, and natural uranium contained in it might reduce the isotope ratio to below 80%; but it should be clearly higher than in soil.

examining the fallout from the Hiroshima event alone. Two studies on soil, rock, and roof tile samples that were preserved in 1945 in Hiroshima itself, and which were retrieved from storage several decades later, exhibited distinct yet very low ^{137}Cs activity [44, 45]. The latter study actually reexamined a series of samples which were reportedly collected by famed nuclear physicist Yoshio Nishina on his visit to Hiroshima only three days after the bombing. Among these, the two samples that had been collected the closest to the hypocenter gave no detectable ^{137}Cs activity. A single sample—obtained from the Koi area, which is located approximately 2 km from the hypocenter and is considered the zone most affected by fallout within the city limits—gave a value of 10.6 mBq/g; all other samples contained less than 1 mBq/g.

Figure 2.3 shows the ɣ-ray spectrum of one of the samples; the ^{137}Cs peak is indicated. Since the measurement was reported in 1996, approximately two thirds of the ^{137}Cs had decayed since the bombing. Most other peaks in the spectrum, particularly ^{40}K, are caused by natural background radioactivity. Concerning this background, Shizuma et al. [45] note:

> *In 1950, soil samples were repacked in air-tight glass vials. … In the present measurement, soil samples were repacked in plastic containers … to eliminate the ^{40}K gamma-ray background from the vial itself.*

Let that sink in for a moment—the radioactivity of fallout from 'Little Boy', collected in the city three days after the bombing, is obscured by that of the glass vials used to preserve it.[5]

Nishina's samples have also been analyzed for uranium isotopes [46]. In this study, the isotope ratio ^{234}U/^{238}U was somewhat variable but always close to 1, whereas the abundance of ^{235}U was consistent with natural background. Therefore, these soil samples, which are untainted by global fallout and very likely were not exposed to rain other than the black rain which transported the fallout,[6] fit into the general pattern of detectable but very low levels of ^{137}Cs, and negligible or absent bomb-derived ^{235}U.

2.3 Cesium and plutonium in soil samples from the Hiroshima fallout area

Low ^{137}Cs activities were also reported by Yamamoto et al. [48], who took samples from soil underneath houses that had been erected throughout the black rain area after the Hiroshima bombing, but before 1950, and thus before

[5] Readers with at least *some* common sense might consider to stop reading here.

[6] The physician Michihiko Hachiya notes in his diary that all days from the 6th to the 9th of August were clear and sunny [47]. It seems possible, however, that some of the sites sampled on the 9th by Nishina were drenched with water before that date by firefighters.

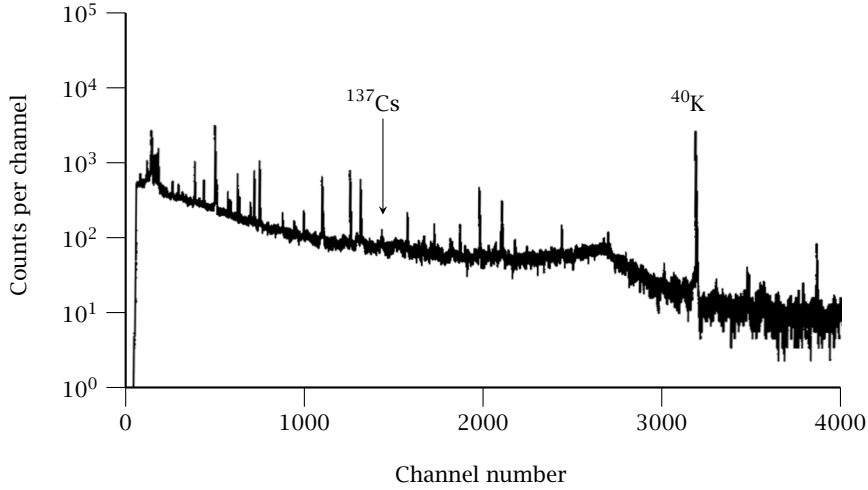


Figure 2.3 ɣ-Ray spectrum measured by Shizuma et al. [45] on one of the samples collected on August 9th by Yoshio Nishina. The ^{137}Cs peak is due to fallout, whereas the ^{40}K peak is part of the natural background.

most of the global fallout struck. All samples contained some ^{137}Cs. The levels scattered by almost two orders of magnitude; however, even the highest values, which were observed in samples from two houses built as early as 1946, remained well below those which are caused in unprotected soil near Hiroshima by the subsequent global fallout. Thus, even in the black rain area, the ^{137}Cs fallout from the Hiroshima bomb was small.

To explain the variability of their observed ^{137}Cs levels, the authors quite plausibly invoke the excavation that may have occurred in preparation for construction in some of the buildings; however, they also state that

> *according to carpenters we interviewed, most of the wooden houses built around this time were built without causing major disturbance of the surface soil,*

which suggests that the fallout may have indeed been quite inhomogeneously distributed within what is considered the fallout area. Conceivably, a variable fraction of the fallout was retained by vegetation cover and prevented from entering the soil.

Disturbed or not before construction, however, the soil should have been protected from any fallout once the houses had been completed. It is remarkable, therefore, that in all of Yamamoto's sub-floor samples *plutonium is also found*. Since the Hiroshima bomb is supposed to have consisted of enriched uranium,

but not plutonium, its fallout should have contained at most minuscule amounts of plutonium.[7]

The observed activity of plutonium (^{239}Pu+^{240}Pu) activity was indeed only about 4% of that of ^{137}Cs (see Figure 2.4B); however, after accounting for the large difference in half-lives, the molar amount of plutonium exceeds that of ^{137}Cs about 20-25 times on average. This ratio pertains to the time of measurement. Plutonium has not decayed significantly since the event, but ^{137}Cs decays much faster and would have been reduced to about one fifth of the original amount between the event and the publication of Yamamoto's study; therefore, the ratio of abundance ($^{Pu}/_{Cs}$) at the time of the bomb drop would have been close to 4.

The authors, starting from the pious assumption that the official story of the bomb is true, stipulate that essentially no plutonium should have been present in pristine samples, and they ascribe that which they find to contamination by the global fallout. Since this completely voids the very premise of their study—namely, that their samples should be free of such pollution—one would expect some effort on their part to explain this unexpected outcome. However, no such explanation is offered. More importantly, the authors do not *test* their assumption that such contamination was possible, which they could have easily done by obtaining soil samples from underneath houses built *before* 1945. If the original premise of the study held, such samples should have been protected from any fallout; on the other hand, according to the authors' revised hypothesis, fallout radioactivity should be present in all of these samples as well.

The only carrier I can think of that might transport some global fallout from soil outside the house to underneath it would be percolating rainwater. Note, however, that according to a preliminary report by the same authors [49] most of the radioactivity was found in a very shallow layer at the very top within the soil (Figure 2.4A). It is difficult to see how percolating water from outside the house would have produced such a distribution. Moreover, plutonium and cesium are not equally mobile within the soil; the aforementioned study by Sakaguchi et al. [42] shows that plutonium is carried downward faster than is cesium, and thus more mobile. Hence, if indeed global fallout had been carried by percolating rainwater from soil outside to that underneath the house, the

[7] A small amount of plutonium would form during the detonation through neutron capture by ^{238}U. From the neutron cross sections for capture and fission of ^{235}U and ^{238}U, their presumed abundances in the bomb, and the fixed abundance of ^{137}Cs among the total fission products, it can be estimated that this ratio should be close to 0.25 only; but the observed ratio is close to 4. Moreover, virtually all of this plutonium should be ^{239}Pu. The fraction of ^{240}Pu, which in the small number of samples thus examined by Yamamoto et al. [48] ranged from 0.13 to 0.19 of the total, is typical of reactor fuel that has already burned up to a considerable degree; however, this much ^{240}Pu would not arise in the detonation of a ^{235}U bomb.

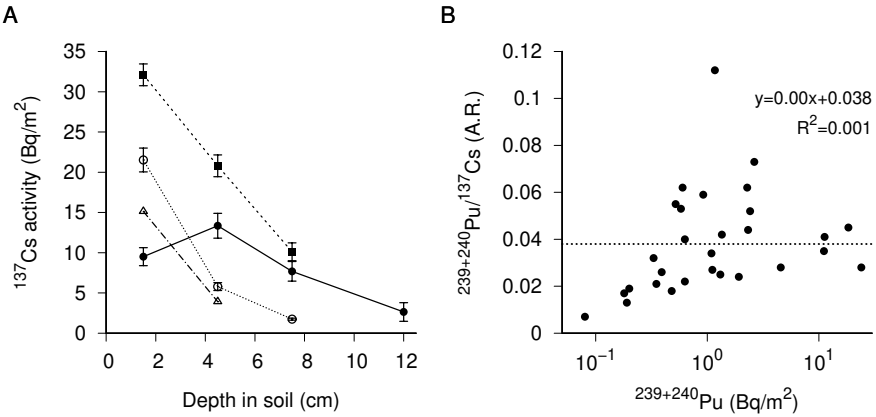


Figure 2.4 Cesium and plutonium activities in soil samples from Hiroshima. A: Activity vs. depth profiles of ^{137}Cs in soil samples retrieved from underneath buildings constructed in the Hiroshima black rain area in 1945-1949. All four individual samples shown in [49] are replotted here. B: Activity ratio (A.R., $^{Pu}/_{Cs}$) in similar samples, grouped by Pu activity. This graph contains all data points from Table 1 in Yamamoto et al. [48]. The equation and R^2 apply to the regression line.

$^{Pu}/_{Cs}$ ratio in the latter place should have been considerably increased. In this case, those among Yamamoto's samples which contain the highest plutonium activity, that is, presumably the highest contamination, should also have the highest ratio of plutonium to cesium activity. However, if we plot the ratio of plutonium activity to cesium activity against plutonium activity, then no such trend is apparent (Figure 2.4B). Thus, percolating rainwater can be dismissed as a mechanism for the presumed contamination.

There is, of course, another explanation for the plutonium in samples that should not have been touched by global fallout—namely, that they *were indeed* not touched by it, and the plutonium was really contained in the fallout of the Hiroshima bomb. This hypothesis has the dual advantage of simplicity and physical plausibility; its only difficulty is that it runs counter to the official narrative. Common sense prefers simplicity; on the other hand, should you prefer your hypotheses bent out of shape in order to circumnavigate taboos, I have another treat for you.

Do you remember that spot of black rain scraped off the plasterboard from Section 1.2? Apart from a whopping 0.2% of bomb uranium, it also contained some ^{137}Cs. The observed ^{137}Cs activity can be compared to that expected from the amount of ^{235}U in the sample attributed to the bomb, the commonly assumed ^{235}U fission yield (about 1 kg out of 50), and the known abundance of ^{137}Cs among the fission products of ^{235}U (approximately 0.06 ^{137}Cs atoms per

^{235}U atom). From such considerations, the authors of that study [5] conclude that ^{137}Cs is eight times *more* abundant in their sample than it should be.

What is their explanation? That somehow the ^{137}Cs became separated from ^{235}U by differential condensation while both were sailing through the air. Thus, in conjunction, the two studies ask us to overlook the separation of cesium and plutonium that slowly, yet inexorably occurs through the persistent percolation of the soil; but on the other hand to assume that efficient separation of cesium from uranium spontaneously does take place in sooty air[8] within the span of only half an hour.

2.4 Cesium and plutonium in the Nishiyama reservoir near Nagasaki

Since the Nagasaki bomb ('Fat Man') used ^{239}Pu, as did most nuclear bombs tested in the subsequent decades, isotopic signatures are less suitable for distinguishing local from global fallout in this case. However, there is one circumstance that makes up for it: at Nagasaki, the heaviest fallout reportedly occurred in and around the Nishiyama reservoir, a small body of water located approximately 3 km from the hypocenter. The timeline of fallout deposition was examined by [50], who analyzed the sediments at the bottom of this reservoir. The lowermost peaks of plutonium and cesium were found at 435-440 cm (Figure 2.5); these must represent the earliest fallout.

The entire sediment core contains only a single layer of macroscopic charcoal particles, which the authors quite plausibly ascribe to the deposition of soot from the burning city. Intriguingly, however, this layer is found at approximately 450 cm. Since the study was published 63 years after the bombings, sedimentation occurred with an average rate of close to 7 cm per year; assuming that this rate was fairly uniform, a separation by 10-15 cm corresponds to a time interval of close to two years.

The authors of the study acknowledge that the peaks are separated, but nevertheless ascribe the radioactivity to the Nagasaki bomb fallout. They do, however, not provide an explanation beyond stating that the mechanism of separation requires 'further study'. Considering the (macroscopic) size of the charcoal particles, we can assume that they are not mobile within the sediment; thus, any separation would have to come about through upward migration of the radioactive isotopes. Such a migration, however, is very unlikely to have happened, for the following reasons:

[8] The black pigment in the rain must have been soot from the burning city, whose houses had mostly been built from wood.

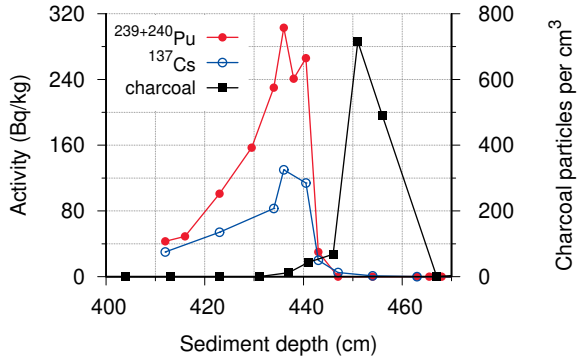


Figure 2.5 Radioactive fallout and macroscopic charcoal particles in sediments from Nishiyama reservoir near Nagasaki. Data excerpted from Table 1 and Figure 2 in Saito-Kokubu et al. [50].

- It lacks a driving force. On dry land, isotopes may slowly be transported downward through the soil by percolating water; however, considering that the reservoir is already water-filled, there will be no upward movement of more water into it from the ground underneath.
- The plutonium and cesium peaks are close to the charcoal layer, but have practically no overlap with it. If the radioactivity had slowly leached out of the charcoal layer, then the radioactive peaks should be broader and exhibit more overlap with the charcoal layer.
- The findings reported by Sakaguchi et al. [42] show that plutonium is carried by percolating water more rapidly than is cesium; therefore, in the reservoir, the plutonium peak should have moved upward further than the cesium peak. However, the peaks of the two isotopes coincide.

As before, there is a politically incorrect but physically straightforward explanation—namely, that the peaks are distinct because radioactivity and the charcoal entered the reservoir at different times, and therefore that the radioactivity is was not delivered by the 'Fat Man'.

Could go into strangely high Pu/Cs ratios here. Also, [51] has some really odd Pu/Sr ratios—^{90}Sr fairly similar between a range of sample locations near the reservoir, Pu much more variable.

2.5 Conclusion

Studies from neither Hiroshima nor Nagasaki furnish any clear evidence of radioactive fallout commensurate with a nuclear detonation. Plutonium levels near Nagasaki are suitably high, but they can't be shown to coincide with the

time of the bombing. The studies on the fallout of the Hiroshima bomb can be summed up as follows:

1. No evidence exists of highly enriched ^{235}U in the fallout. The measurements on soil or on black rain drops dried *in situ* report a very low degree of isotopic enrichment only. A high degree of enrichment is only ever *stipulated*, and the calculations based on this premise result in vanishingly small absolute amounts of bomb uranium.
2. ^{137}Cs attributable to the Hiroshima bomb is readily detected. Its level remains well below the global fallout that arose from later bomb tests, but it nevertheless exceeds the amount we should expect from ^{235}U measurements in conjunction with the key tenets of the official story of the bomb.
3. Samples protected from global fallout also contain plutonium, in amounts and isotopic compositions that are incompatible with its formation by a detonating ^{235}U bomb.

All of these observations are consistent with a dirty bomb containing reactor waste. Nuclear reactors mostly run on uranium enriched to only about 3% of ^{235}U. Of all the ^{235}U nuclei undergoing fission in the reactor, approximately 6% produce ^{137}Cs as one of their fission products. Since ^{238}U is highly abundant in the fuel, a significant proportion of the neutrons released by ^{235}U fission is captured by ^{238}U to form ^{239}Pu, which can in turn capture a second neutron to become ^{240}Pu. The ratio of plutonium thus generated to uranium fissioned is known as the *breeding rate*. This rate can approach or even exceed unity, although it was likely lower in those early reactors.

From the ratio of ^{137}Cs to plutonium in Yamamoto's study [48], we can estimate that, in the history of this fallout nuclide mixture, approximately four times more nuclei have undergone fission than are now present in the form of plutonium. This gives a minimum breeding rate of 0.25; considering that plutonium, once formed, may also fission, the rate was likely somewhat higher. A number of this magnitude fits with at least partially spent reactor fuel, as does the ratio of ^{240}Pu to ^{239}Pu (see footnote on page 25 above).

What is the ratio of ^{137}Cs to ^{235}U in the fallout? Shizuma et al. [5] concluded that ^{137}Cs was present in eightfold excess over ^{235}U in their sample, but that estimate presupposed a high degree of ^{235}U enrichment, and also a low fission yield. If we assume that the ^{235}U in 'Little Boy' was enriched to only 3% rather than 80%, then the fraction of ^{235}U in Shizuma's samples which we should attribute to the bomb rises in inverse proportion; thus, we now have a threefold excess of ^{235}U over ^{137}Cs at the bomb's official low fission yield. To bring the

proportion back into line, we have to reduce the fission yield from the official 2% to less than 1%.

We therefore have a low fission yield reflected in the ratio of the starting material (^{235}U) to the fission product (^{137}Cs), and a high fission yield indicated by the presence of plutonium (particularly ^{240}Pu). To reconcile these two findings, we must assume that the dirty bomb, in addition to some reactor waste, was also laced with some pristine reactor grade uranium that never had gone critical. Of course, unlike highly enriched uranium (see below), both types of material were readily available; thus, our interpretation poses no practical difficulty.

2.6 Addendum: enrichment of uranium to bomb grade—was it feasible in 1945?

As mentioned before, natural uranium contains only 0.72% of the fissile isotope ^{235}U, which is said to have been concentrated to 80% in the bomb fuel, of which the bomb contained approximately 50 kg. The methodology is said to have combined gas diffusion and electromagnetic separation. For gas diffusion, the uranium is converted to a volatile compound (UF_6). The gas molecules that contain the lighter isotope will move through space slightly faster, which can be used for their selective capture. However, performed just once, the process achieves only a very slight degree of enrichment, and it thus has to be repeated very many times, with each successive step increasing purity but also decreasing yield. Conversely, electromagnetic separation is highly effective in principle, but only on a very limited scale.[9]

How far along was the technical realization of isotope separation before the bomb was allegedly completed? Glenmore Trenear-Harvey in his book *Historical Dictionary of Atomic Espionage* [52] quotes from a conversation between the physicist Klaus Fuchs, a member of the Manhattan project and also a Soviet spy, with his spy handler Harry Gold from February 5th, 1944:

> *The work involves mainly separating the isotopes ... should the diffusion method prove successful, it will be used as a preliminary step in the separation, with the final work being done by the electronic method. They hope to have the electronic method ready early in 1945 and the diffusion method in July 1945, but K [Fuchs] says that the latter estimate is optimistic.*

[9] Even assuming sufficient separation in a single step, this method would still have required that $50\text{kg} \times {}^{80\%}/_{0.72\%} = 5.55$ tons of raw uranium be shot through what is essentially a mass spectrometer. That seems like a tall ~~tale~~ order.

Again according to Trenaer-Harvey, Fuchs met with another spy handler, Stepan Apresyan, in June 1944 and reported that

> *... the ISLANDERS [British] and the TOWNSMEN [Americans] have finally fallen out as a result of the delay in research work on diffusion.*

Fuchs continued working for the Soviets throughout the war and afterwards, but he never could give them a description of a viable enrichment process. This is apparent from the technical development pursued during the late 1940s and early 1950s by the Soviets themselves. The German physicist Max Steenbeck, who played a leading role in this effort, gives a first-hand account of it in his autobiography [53]. Before the experimental work began, the Soviets conducted broad consultations to identify the most promising physical principles of separation, and indeed there were some false starts before the successful development of the gas centrifuge. Thus, even though the Soviets had supposedly come into possession of America's most prized atomic secrets, clearly those secret files did not tell them how to enrich ^{235}U.

Steenbeck, who had himself been kidnapped by the Soviets as a civilian in Berlin, recruited several German and Austrian scientists and technicians from Soviet POW camps; two of them, Zippe and Scheffel, stayed and worked with him throughout his whole time in the Soviet Union. When finally all three men were allowed to return to Germany in the mid-1950s, Steenbeck joined his family in Jena in East Germany, whereas Zippe and Scheffel settled in the West. They were snapped up by Degussa, a metallurgical company with interests in nuclear fuel, for which they implemented the gas centrifugation technique on an industrial scale. Evidently, there was at the time no better or equally good process in place at this leading Western company. Centrifugation quickly superseded all other techniques for industrial ^{235}U enrichment and remains the standard method today.

Overall, this bit of history strongly suggests that the technology for enriching uranium to bomb grade, in quantity, did not exist in 1945. It may not exist even now, for even if feasible it would seem pointless, since it certainly is much easier to enrich plutonium, which can be bred from ^{238}U in nuclear reactors and then separated from the starting material using chemical methods.[10] Moreover, since ^{239}Pu has a much lower critical mass, the amounts required are far smaller than

[10] Steenbeck also states that the highest degree of ^{235}U enrichment he achieved by centrifugation was 30%. That is well above requirements for reactor fuel, yet it falls short of the 80% that was allegedly achieved (and required) for 'Little Boy'. It is claimed, however, that the 'Health Physics Research Reactor' used in 1961-62 during 'Operation Bren' to mimic the spectra of ɣ-rays and neutrons produced by the Hiroshima bomb contained ^{235}U enriched to 93% [33].

the ^{235}U required for a uranium bomb. Plutonium was purportedly used for the 'Fat Man' bomb in Nagasaki, and to my knowledge for all fission bombs thereafter.

Even if we assume that the United States did at the time have the capacity to produce bomb grade uranium, doing so would have diverted partially enriched ^{235}U from the use as reactor fuel, which was in turn required to make plutonium. Pursuing uranium and plutonium bombs at the same time would have hampered both; there would have been a strong incentive to prioritize either one.[11]

In summary, feasibility arguments reinforce the conclusion reached by the analysis of fallout studies, namely, that 'Little Boy' did not contain highly enriched uranium—and thus, of course, could not have produced a nuclear detonation. With that in mind, let us now consider some of the physical evidence adduced to prove that such a detonation did indeed occur.

[11] Just consider those numbers again—only about 1 kg out of 'Little Boy's' 50 kg highly enriched uranium is said to have fissioned. Considering how hard it is to make, would that seem like a good use of the material?

3. Early measurements of residual radioactivity

As explained in Section A.10, most of the radiation produced by a nuclear bomb is released at the time of the detonation in the form of ɣ-rays and neutrons. Both can in principle be monitored in real time with suitable detectors [33], and the means were already available in 1945. The ɣ-radiation, in particular, should have been picked up by X-ray dosimeters, of which several types were already known in the 1940s [54], and at least the more modern hospitals in Hiroshima should have been equipped with them. I have not seen any reports of X-ray dosimeter readings that were taken during the bombing, but of course at that instant nobody had reason to suspect that an atomic bomb had been dropped. The upshot is that we have no record of an immediate, quantitative measurement of the radiation released during the blast.

In the absence of such direct measurements, one can try to reconstruct the radiation intensity during the detonation from indirect measurements of induced radioactivity (Section A.10.3) and of thermoluminescence (Section A.8.3). Here, we will consider such measurements that occurred on-site in the days and weeks directly after the bombing. These measurements uses Geiger counters or similar devices that could not identify radioactive isotopes, which also implies that they could not distinguish between the fission products carried by radioactive fallout and induced radioactivity. They are nevertheless of great value, since both fallout and induced radioactivity comprise mixtures of isotopes with very different half-lives. The very short-lived isotopes would produce high intensity beginning with the detonation but would drop to insignificant levels after time intervals ranging from hours to weeks; thereafter, the much slower decay of the longer-lived isotopes would sustain a residual activity somewhat above the natural background for several months to years. Thus, a high initial level of radioactivity which then rapidly drops by several orders of magnitude would be characteristic of a nuclear detonation. On the other hand, absence of the initial short phase of high activity would indicate that no such detonation had occurred.

3.1 Timeline and findings of early field measurements

Given the great potential value of early measurements, there is a striking shortage of actual data. The ^{235}U bomb supposedly dropped upon Hiroshima had never been tested before, and has never been used again. Under these circumstances, one surely would expect that the Americans would have started their investigations at the earliest opportunity after the Japanese surrender; in fact, already *before* the surrender they might have advised the Japanese of the best ways to ascertain the nature and effect of the weapon. They might even have asked a neutral third party to assist the Japanese with the investigation, which would have been in the best interest of both sides. However, it seems the Japanese received no such assistance. Even more strikingly, after the capitulation it still took the Americans several weeks to send even some small advance parties of investigators; not before October did the Manhattan Engineers begin their own measurements (see Table 3.1). Neither did they make up for lost time afterwards. The American physicist Robert Wilson, writing on the bomb radiation in 1951, began by summing up the state of this research [55]:[1]

> *It is no simple problem to determine the X-ray and neutron dosages which were received at Hiroshima and Nagasaki. Much of the meager primary data that were written down has been dispersed or lost—that which existed but was not written down is mainly forgotten.*

A timeline of early measurements, by both Japanese and American investigators, is given by Imanaka [57]. Table 3.1 provides a summary. While there is some variation in the results, all measurements agree that the level of activity is above the natural background but overall quite low, certainly nowhere near the levels required to induce acute radiation sickness xref. Among these results, the most important are those of August 11$^{\text{th}}$, since they were obtained just five days after the bombing, and thus within a time period during which there should still have been substantial activity from short-lived isotopes.[2] This is illustrated in Figure 3.1A, which shows the level of induced radioactivity at the hypocenter

[1] Wilson's paper was published in 1956, but a footnote states that it was written in 1951 at the request of the Atomic Bomb Casualty Commission and declassified only in 1955. The paper repeatedly advises the reader that, due to the shortage of empirical data, the conclusions of this paper should be taken as educated guesses only. Wilson cites all of six references, which illustrates his limited access to information. I unsuccessfully tried to obtain one of these [56]—it remains in the poison cabinet to this day.

[2] The only report I have found of an even earlier measurement is that by Toland [58], who states that Dr. Fumio Shigeto, then vice director of the Hiroshima Red Cross hospital, used an X-ray dosimeter to detect radiation at the hospital on the day after the bombing (August 7$^{\text{th}}$) and found very little.

Table 3.1 Early measurements of environmental radioactivity in Hiroshima. Excerpted from Imanaka [57].

Team/University	Date	Location and findings
Osaka	August 11th	up to five times natural background several hundred meters from the hypocenter
Kyoto	August 11th	up to 10 times background several hundred meters from hypocenter
Kyoto	August 15th/16th	six times natural background at Asahi bridge, otherwise weak activities
RIKEN	August 17th to October 20th	[57] reports only relative readings; values in August and October of similar magnitude
RIKEN	September	activity up to 6 times above background in fallout area
RIKEN	October 1-22	activity up to 9 times above background near hypocenter
RIKEN	January 1946	activity near hypocenter 6 times above background, fallout area 3 times background
Manhattan Engineers	October 1945	activity up to 15 times above background near hypocenter, up to 8 times in fallout area
Hiroshima	1948	up to 2.5 times background in fallout area

as a function of time for the first three months after the bombing. The shape of the curve has been inferred from later experiments, in which soil samples from Hiroshima were irradiated with neutrons, and the activities of the major isotopes produced by neutron capture were measured.[3] The height of the curve was calibrated to a single reported measurement, which was taken 87 days after the bombing; according to Ishikawa et al. [7], and in keeping with the general trend evident from Table 3.1, this measured value amounted to ten times the natural background.

Toland [58] and Liebow [59] also report that X-ray film stored in sealed packages within the same hospital was blackened after the bombing. This observation is often cited as evidence of ionizing radiation released in the blast, but while it may have prompted Dr. Shigeto's measurements, the negative outcome of the latter suggests that the films may have been blackened e.g. by exposure to heat when the hospital was burning.

[3]This graph was produced using the data in Table 9 in Okajima et al. [25], on which those authors also base their own 'official' estimate of induced radiation dosage. The single measured data point used to scale the curve is also given in that reference. A very similar graph appears in Figure 5-2 of Ishikawa et al. [7].

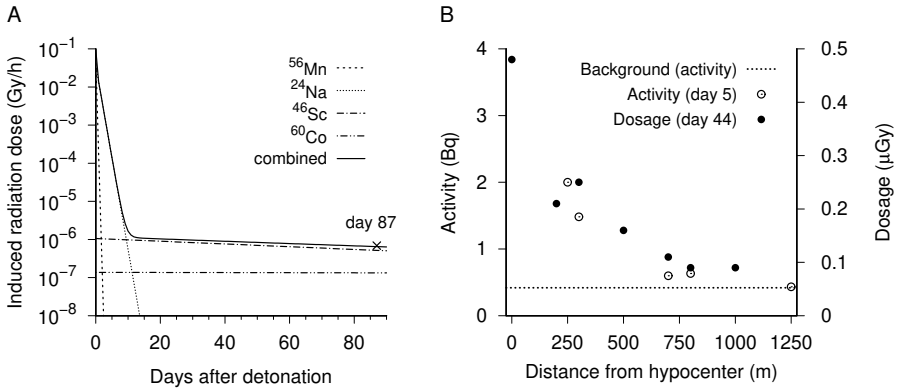


Figure 3.1 Estimates and measurements of induced radioactivity in Hiroshima. A: Induced radioactivity at the hypocenter as a function of time, extrapolated from a single measured data point (day 87) according to Okajima et al. [25]. The solid line is the sum of all individual isotope activities. See text for further explanation. B: Two data series shown by Takeshita [60]. The measurements after 5 days are given in Bq, whereas those on day 44 are given in µGy.

It is evident that the estimated activity changes very little after the 15$^{\text{th}}$ day. On the other hand, measurements within the first week should have shown a much higher activity. The question then is: did they? It seems that activity at the hypocenter was not measured within that time frame,[4] however, we can estimate it by comparing the two data series shown in Figure 3.1B. These data were collected 5 days and 44 days, respectively, after the detonation. The measurement on day 5 is scaled in units of activity (Bq, or decays per second), while the measurement on day 44 is given in units of absorbed energy dose (µGy).[5] They have been overlaid and scaled to show that they vary similarly with the distance from the hypocenter, as they should. We can use this similarity to estimate that the activity on day 5 at the hypocenter would have been approximately 4 Bq, which is some ten times above the natural background. The single measured data point in panel A, at 87 days, was also about 10 times above the natural background [7]. Thus, while the neutron activation experiment shown in panel A indicated that between day 5 and day 87 radioactivity should have decreased by a factor of 100, the observed factor is 1—that is, no decrease has occurred. Even

[4] It is not clear who first determined the location of the hypocenter, or when; but in all likelihood it was not known or agreed upon at such a short time after the bombing.

[5] This plot combines panels A and B from Figure 1 in Takeshita [60], with units of measurement converted to the ones preferred in this text. The first data series was obtained on August 11$^{\text{th}}$ by a team from Osaka University. The second data series was likely obtained by researchers from RIKEN, but I have found no English-language reference to confirm this explicitly.

though both of these factors are approximations, they cannot be reconciled; one must be false.

3.2 Shimizu's sulfur activation measurements

Against the various accounts of weak observed activity throughout the early period, one report stands out—that by Sakae Shimizu [34], one of the researchers from Kyoto University who undertook several expeditions to Hiroshima in August and September (see Table 3.1). The key pieces of his evidence are a magnetic piece of iron, a horse bone, and three porcelain insulators containing sulfur. When these samples were examined for β-radiation in the laboratory at Kyoto, all showed significant activity, which was ascribed to activation by neutrons. Of particular interest is the activation of sulfur, since it requires highly energetic (fast) neutrons [33], which unlike those of low energy would be expected in a nuclear bomb but not in the natural background radiation.

There are several reasons, however, to view this evidence with some suspicion. Most importantly, it was not pursued any further. Activation of sulfur (^{32}S) would have been singularly useful to determine the strength and exact location of the detonation, as well as the reach of the fast neutrons produced by it. In addition to being selective for fast neutrons from the bomb, sulfur is quite abundant. After the bombing, those ceramic insulators must have been lying on the ground near toppled and burned power poles everywhere, and plaster from buildings would have been another plentiful source of sulfur. The activation of sulfur produces radioactive phosphorus (^{32}P), which has a half-life of 14.3 days. Thus, if Shimizu's early high readings were correct, enough activity should have remained even at 4-6 weeks after the detonation, that is, long enough for the Americans to carry out their own measurements. There is, however, no indication that they ever did so.[6]

One must also note that ^{32}P can be produced from two precursors—from ^{32}S by capture of fast neutrons, and from ^{31}P by capture of slow neutrons. Thus, the mere detection of ^{32}P is not enough; the ambiguity concerning its origin must be resolved by determining the sample's content of both precursor isotopes through chemical analysis. No such analysis is reported in [34].

[6]Instead of following Shimizu's lead, his American handlers confiscated all of his written records and then 'lost' them (see Section 1.5.5). As another interesting aside, Shimizu [34] also notes: "Due to physical fatigue and may be to an effect of exposure to nuclear radiations during the field survey in Hiroshima, in the night of the 19th I spat out much bloody sputa, and I was forced to lie on a bed for about three months." Neither fatigue nor the weak radioactivity on the ground in Hiroshima could account for Shimizu's hemoptysis (coughing of blood); however, exposure to mustard gas very well could.

In the end, however, Shimizu's report should not be singled out for lack of experimental detail. Similar objections could be raised against the other listed studies, at least as far as they are available to us; and if we want to judge impartially, our choice is either to take them all at face value or to disregard the lot. If we accept them, we are faced with conflicting evidence. Among the measurements on the ground, only those that occurred in the first week have any real power to confirm or refute a nuclear detonation, and they refute it; on the other hand, Shimizu's laboratory findings on sulfur activation would confirm it.

The overriding impression one gathers from this unsatisfying state of affairs is simply that which was already spelled out by Wilson [55], namely, that both the acquisition and the documentation of early radioactivity measurements were wholly inadequate; and this inadequacy speaks louder than the evidence itself. If the official story had been true, if the bomb had indeed been the world's first and only ^{235}U bomb, such obviously willful negligence would be inexplicable. Fantastic amounts of work and treasure had been poured into the development of this revolutionary weapon; surely those who had accomplished it would also want a detailed record of the outcome and proof of their success. If, on the other hand, the official story were indeed a lie, then the neglect would be entirely understandable, since richer and more detailed 'evidence' would only increase the chances that the fraud might be uncovered in the end.

4. γ-Ray dosimetry by thermoluminescence

When a fission bomb detonates, radiation exposure will be highest at the *epicenter* of the detonation in the air; of all places on the ground, the *hypocenter*, that is, the spot vertically underneath the epicenter, will receive the highest dosage. With increasing distance from the hypocenter, the radiation dosage will decrease rapidly; and at any given distance, it may be reduced through *shielding* by concrete buildings or other structures.

Both γ-rays and neutrons can in principle be monitored promptly with suitable detectors [33], and the means were already available in 1945. When such direct readings are lacking, as is the case in Hiroshima and Nagasaki, one can still try to determine in hindsight how much neutron and γ-radiation was released in the burst. For γ-rays, this can be done through thermoluminescence measurements on suitable rocks or ceramics that were exposed during the blast; the neutron radiation can be quantified from induced radioactivity. Measurements of this kind are indeed the showpieces among the evidence advanced to prop up the official story; and taken at face value, their findings leave no other conclusion than that some sort of nuclear detonation must indeed have occurred. We will consider both methods and their applications in turn, beginning in this chapter with thermoluminescence. We will focus on two early studies which used thermoluminescence measurements on bricks or tiles to determine the γ-dosages that were in Hiroshima and Nagasaki [61, 62]. The procedures used by both are similar in principle, but they show surprising differences in detail that highlight significant pitfalls of both studies (see Table 4.1).

4.1 Calibration of thermoluminescence measurements

As explained in a little more detail in Section A.8.3, the term refers to the observation of light given off by ceramic materials when these are heated up gradually; the intensity of the light thus evoked is proportional to the dose of γ-radiation which this material was exposed to earlier, and potentially a very long time ago.

Table 4.1 Thermoluminescence measurements on tiles and bricks in Hiroshima and Nagasaki: comparison of assumptions and findings reported by two early studies.

Study	Higashimura et al. [61]	Hashizume et al. [62]
Use of roof tiles from wooden buildings	suitable and used as samples	not usable, since orientation relative to hypocenter unknown
Calibration	irradiation with ^{60}Co	'spectrally equivalent' combination of X-rays, ^{60}Co, and ^{137}Cs
Glow curve shape	bomb and calibration samples are different	bomb and calibration samples are similar
Thermoluminescence signal at 180°C	not detectable in bomb-exposed tiles	detectable with lifetime of 6.7×10^5 years, used exclusively
Thermoluminescence signal at 330°C	detectable with lifetime of 100 years, used exclusively	not used
Possible loss of signal due to fire	considered, said to be avoided by sample selection	not mentioned
Possible loss of signal due to bomb flash	not mentioned	not mentioned
Depth distribution of signal in sample	not mentioned	determined only on a single calibration sample

A crucial step in this procedure is to establish the relation between the activating γ-ray dose and resulting thermoluminescence intensity. This relation will be affected by the chemical composition of a particular piece of ceramic, and therefore the measurement must be calibrated empirically for each sample. To this end, both studies use the same clever trick: they first heat the brick or tile in question to obtain an uncalibrated measurement of the thermoluminescence originating from the bomb. This heating run will purge the material of all pent-up thermoluminescence. The deactivated material is then recharged by irradiating it with a known dose of γ-radiation from a laboratory source. From the amount of light released when the sample is heated again, the dose-response proportionality can be determined and used to calculate the γ-dose that would have caused the thermoluminescence signal which was measured first.

Unless proven otherwise, one must assume that the efficiency of activation may vary with the energy of the impinging γ-particles. To account for this, Hashizume et al. [62] employ a combination of various sources claimed to match

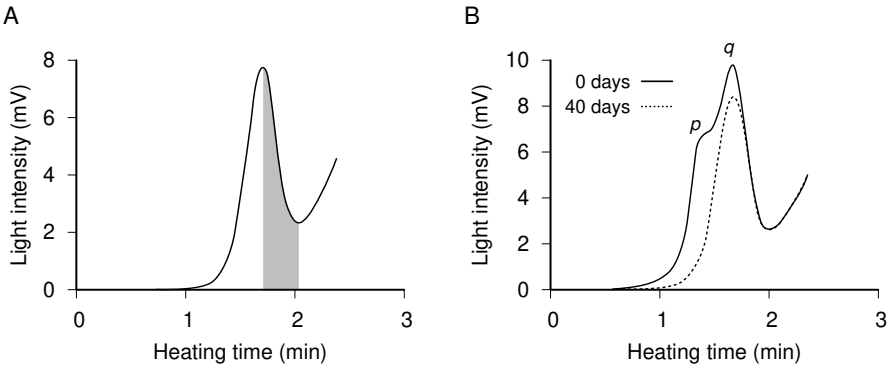


Figure 4.1 Thermoluminescence curves of brick or tile samples, drawn after Figure 6 (A) and Figure 10 (B) in Hashizume et al. [62]. Samples were heated from ambient temperature to 300°C within 3 min. A: Described as 'a typical glow curve of thermoluminescence', which was 'obtained from a sample.' The portion of the signal curve indicated by the shaded area was used to determine the absorbed ɣ-ray dose. B: Glow curve of an experimentally irradiated sample, showing two overlapping peaks at approximately 1.4 min/140°C (p) and 1.8 min/180°C (q), respectively. 40 days after irradiation, p has vanished, whereas q persists; with this information in hand, Hashizume et al. estimate its life time at 670,000 years.

the energy spectrum the bomb radiation.[1] In contrast, Higashimura et al. [61] employ only a single ^{60}Co source.

Taken at face value, the calibration procedure adopted by Hashizume et al. [62] would seem superior. There is, however, serious cause to doubt their assertions. In one of their experiments, Hashizume slice up a brick into layers of 1 cm thickness to determine the depth distribution of thermoluminescence.[2] The result is reproduced here in Figure 4.2A. Now, this depth distribution would depend on the energy spectrum of the activating ɣ-radiation, since softer (i.e. lower-energy) radiation would exhaust itself closer to the surface, while harder rays would penetrate and cause activation in deeper layers also. Thus, this experiment would be a good way to validate the authors' assumptions about the bomb energy spectrum, and furthermore to observe changes to this spectrum with increasing distance from the hypocenter. It is very strange, therefore, that this experiment was carried out only once, and *only on an experimentally irradiated sample, but never on a native one.* This is just one of several issues

[1] The sources used by Hashizume et al. [62] were ^{60}Co, ^{137}Cs, and a linear accelerator producing high-energy X-rays, which differ from ɣ-rays only in origin but not in nature. The proportions and the X-ray energies are not given, and the assumed bomb ɣ-spectrum is not detailed either.

[2] The authors do not detail which, if any, precautions were taken to avoid heating of the brick when it was cut, which might trigger and deplete the thermoluminescence prematurely.

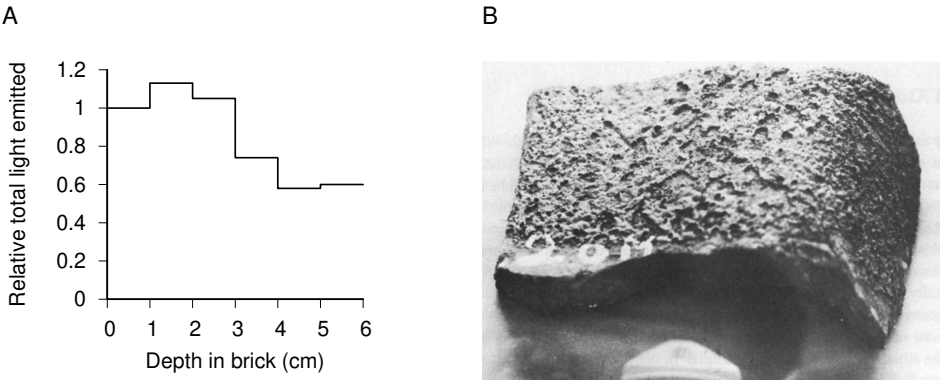


Figure 4.2 Depth profile of thermoluminescence intensity in a laboratory-irradiated brick, and roof tile from Nagasaki with surface damaged by heat. A: The brick was exposed to ɣ-rays, cut into layers, and the thermoluminescence intensity of each layer was measured separately. Replotted from Figure 7 in Hashizume et al. [62]. B: Bubbled and roughened surface of a roof tile found in Nagasaki. Photograph taken from Ishikawa et al. [7], who assert that the observed effects are due to the flash of light from the bomb.

that raise the question how this entire study could possibly have survived serious peer review.

4.2 Signal shape and stability

Another flaw in Hashizume's study is their failure to clearly identify any of the few glow curves they show as that of a native sample rather than a calibration run.[3] The authors do suggest that native and calibration signals are similar in shape, but they never prove it. The signal shown in their Figure 6 (reproduced here as Figure 4.1A) is referred to as "a typical glow curve from a sample," which is suggestive yet remains ambiguous; all other data shown are described as originating from laboratory activation. Showing some native and calibration runs side by side would have greatly helped their case, and it is difficult to imagine that none of our fearless yet imaginary peer reviewers would have demanded it.

Another questionable feature is the assumed stability of the thermoluminescence signal in Hashizume's selected temperature range. While the x axes in Figure 4.1 are labeled in units of time, the rate of heating to the final temperature of 300°C at 3 min would have been fairly linear, and the two overlapping peaks in panel B would be located at approximately 140°C and 180°C, respectively.

[3] On a related note, Higashimura et al. [61] do not show any raw data at all, which considering the novelty of their study is highly unusual.

The temperature at which a given luminescence peak occurs correlates with the *activation energy*, that is, the height of the energy threshold that the trapped electrons in the sample must overcome in order to return to what is ultimately a lower state of energy. This also translates into different stability at ambient conditions; the lower the trigger temperature, the more readily the peak will fade over time even without any heating of the sample.

Hashizume et al. [62] report that their lower-temperature peak (labeled with *p* in the figure) disappears within 40 days of experimental irradiation, but claim that the other one (peak *q*) should be stable with a lifetime of 6.7×10^5 years.[4] Accordingly, they use the right half of this peak to quantify the radiation dose in all their samples (cf. Figure 4.1A). However, such an enormous difference in lifetime for peaks that are separated by only some 40°C seems unlikely. Indeed, a very different assessment is given by Higashimura et al. [61], who report that in their bomb-irradiated samples no peak at 180°C is observed, although it does occur after experimental irradiation with ^{60}Co:

> *Glow curves resulting from bomb radiation in the past and from the ^{60}Co irradiation in the present are different in shape. The glow curve resulting from ^{60}Co irradiation shows … a distinct peak at about 180° C. On the contrary, the glow curve resulting from bomb radiation has a negligible intensity below 180° C.*

Accordingly, they discard the peak at 180°C altogether and instead evaluate only the signal evoked at a much higher temperature range (≥ 330°C), for which they nevertheless much more modestly claim a lifetime of "longer than 100 years." In summary, between the failure to clearly show that any of their own bomb-exposed samples exhibit this peak, and the starkly contrasting observations from the earlier study [61], the findings reported by Hashizume et al. [62] cannot be trusted.

4.3 Sample inactivation by heat from the bomb and the fire

If bricks and tiles were to be used for retrospective evaluation of thermoluminescence, it was essential that they be kept at gentle temperatures throughout, from the moment of their activation by the blast to the laboratory measurement. Premature thermal inactivation could have occurred either due to the heat from the bomb itself, or from the subsequent fires. Higashimura et al. [61] state that

[4] The lifetime of an exponential decay (as will be assumed with a fading process such as this) is defined as the time within which the original signal decays to a residue of $1/e$ (approximately 0.37). The stated lifetime corresponds to a half-life of 4.64×10^5 years, which is roughly equivalent to 4 successive ice age cycles.

they used roof tiles which came from areas that were not affected by the fire. However, such areas must have been very hard to find. In their book chapter on the extent of the fires in Hiroshima that followed the bomb attack, Kawano et al. [63] state:

> *Within 30 minutes after the bomb blast, large fires broke out and firestorms started. After 2 to 3 hours, the wind speed had reached 18 m/s, and from 11:00 [11 a.m.] to 15:00 [3 p.m.] fierce whirlwinds occurred in some areas in the northern half of the city. The winds subsided at about 17:00 [5 p.m.] that afternoon. As a result of the firestorms, anything that was burnable was completely destroyed in an approximately 2 km radius from the hypocenter.*

'Anything burnable' should certainly include the wooden buildings whose roof tiles were used for Higashimura's study; all of their samples are said to have been collected within 1 km of the hypocenter.[5] Indeed, Hashizume et al. [62] forgo those roof tiles altogether. Ostensibly, however, this is not because of the direct effect of the fire, which they avoid to discuss altogether, but for a more fastidious reason: since all those wooden houses had been 'destroyed,' it was no longer possible to tell how the roof tile in question had been oriented relative to the impinging ɣ-rays. This unknown angle would have affected the absorbed dose and thus have been a source of significant yet unaccountable variation. To solve this relatively minor problem, they restrict themselves to flat tiles and bricks from concrete buildings that had been left standing after the attack, and for which the orientation toward the center of the detonation was therefore known. They also emphasize that all their samples had been in a direct line of sight to the center of the detonation, and therefore received an unshielded dose of ɣ-rays.

How does Hashizume's choice of samples affect the question of thermal inactivation? While many large buildings were left standing after the attack, they also were affected by the fire. In the evening of August 8th, that is, two and a half days after the detonation, the physician Michihiko Hachiya noted in his diary [47]:

> *Concrete buildings near the center of the city, still afire on the inside, made eerie silhouettes against the night sky. The glowing ruins and the blazing funeral pyres set me wondering if Pompeii had not looked like this during its last days.*

[5] Even if those tiles looked undamaged by the fire, they still might have been thermally inactivated, since this will occur at lower temperatures than those required to mar the surface.

Figure 4.3 Three of many burnt-out buildings that according to various studies [64, 65] yielded pristine tiles or bricks suitable for measurement of ɣ-ray dosage by thermoluminescence. Top: Hiroshima City Hall; center: Hiroshima Prefectural Industrial Promotion Hall (now commonly called the 'Atomic Bomb Dome'); bottom: Shiroyama elementary school in Nagasaki.

The impressions conveyed by this quote and by that of Kawano et al. [63] given above certainly mirror those one gleans from photographs of the scorched and destroyed city. The examples in Figure 4.3 show three buildings from which samples were obtained that were allegedly used with success for thermoluminescence measurements.[6] The fires that left their marks on these buildings broke out some time after the detonation, that is, after the bricks and tiles would have had received their dose of γ-rays and been activated for thermoluminescence. Now, maybe we can't be absolutely sure that *every single brick or tile* from such a building would have been thermally discharged in the conflagration; but at the very least, a lot of them must have been, and thus a very large proportion of duds would have been among the samples later collected from these buildings for thermoluminescence measurements.

A related problem arises in connection with the intense flash of light and thermal energy released by the detonation. The heat is said to have etched the surfaces of unshaded granite tombstones, and so reliably and regularly that from the outlines of the shadows thus produced the epicenters of the explosions in both cities could be determined with high accuracy (see for example Hubbell et al. [66]). Figure 4.2B shows a a roof tile which was collected at 270 m from the hypocenter in Nagasaki, and whose surface corrosion is portrayed as the direct effect of the thermal flash [7]. If this is true, then several of Hashizume's samples, which were collected at similar or even shorter distances from the hypocenters in both cities, should have shown similar thermal damage to the surface; for as the authors insist, the samples were exposed to the γ-rays without obstruction, and therefore also to the thermal flash.

Considering that such damage only occurs at temperatures substantially higher than those used in their thermoluminescence experiments, it will have occurred to them that thermal inactivation must at the very least be *considered* and measured in control experiments. They already had found a technique that would suit this purpose, namely, the comparison of thermoluminescence in superficial layers to that in deeper ones (see Figure 4.2A). That they do not even *mention* the problem means that their work is unreliable; and so is any such study that does not admit to and convincingly address the problem of thermal sample inactivation. So far I have found not one study that clears this bar.[7]

[6] These particular buildings are listed in Ichikawa et al. [64] and Egbert and Kerr [65]. Hashizume et al. [62] only give latitudes and longitudes for the locations of their samples; none of these coincide with any of the landmark buildings that one finds depicted and identified in photographs, but one pair of coordinates points to water in a river arm, and another one to a spot of wilderness far from the city.

[7] Ichikawa et al. [64], in another experimental study on roof tiles, state that "although the roof tiles were collected with much care to obtain samples which had not suffered from the fire, some

4.4 Appraisal of reported luminescence data

Higashimura et al. [61] report only the final numbers in terms of ɣ-dose at different distances from the hypocenter, so the reader is given no opportunity to judge the actual experimental data obtained by these authors. However, the more detailed study by Hashizume et al. [62], while also showing only very few of its raw glow curves, does give the luminescence intensities determined from them (see their Table 2). It also gives the formula used to calculate the ɣ-ray dose from the luminescence values:

$$\gamma\text{-Dose} = L \times G \times C \times R \tag{4.1}$$

In this equation, L is the bomb-induced thermoluminescence measured in the first heating run on each sample, whereas G, C, and R are calibration and correction factors. The most important one of these is G, the calibration factor that gives the amount of ɣ-rays required to induce a certain luminescence response (γ/L), as determined from the second heating run. C is a factor that corrects for the orientation *in situ* of each sample relative to the incident ɣ-rays; this number varies only from 1.09 to 1.31 and thus has a minor effect on the overall result. R is supposed to correct for fading between the times of activation and of measurement; no value is given for it, but using the very long lifetime which the authors assume for the luminescence peak q (see Table 4.1) its value will be very close to 1.

With this in mind, one surely would expect any major change in the ɣ-dose to correlate with major changes in L also, which therefore should assume its highest value near the hypocenter, while variations due to the correction factors should be relatively minor. This is, however, not what we find. Figure 4.4A shows Hashizume's data from Hiroshima. We see that the raw data for L vary only slightly, and in fact reach their highest value at the greatest distance from the hypocenter. Nevertheless, a strong and regular decreasing trend is shown for the ɣ-dose, which is entirely due to a closely similar trend in the γ/L calibration factors.

After recovering from the surprise, we might wonder if it is *physically* plausible that bricks and tiles, which likely are quite similar in chemical composition, should show such large variation in their sensitivity to activation by ɣ-rays. This

samples did not show any thermoluminescence, which probably reflected the fire effect. But since we took only the glow curves of the normal type …" While this explanation is of course much better than nothing at all, it does not address possible *partial* thermal inactivation. Moreover, this paper explicitly lists several burnt-out or burnt-down buildings among its sampling sites, including Shiroyama school in Nagasaki (see Figure 4.3) and Hiroshima Castle, of which reportedly [67] only the foundation walls had survived the bombing.

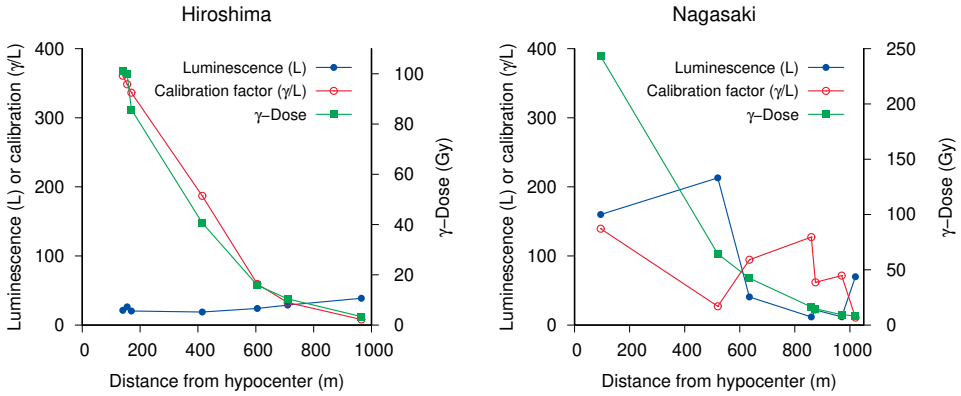


Figure 4.4 Sample thermoluminescence, calibration factors, and ɣ-dosages as functions of distance from the hypocenters in Hiroshima and Nagasaki. All data are from Table 2 in Hashizume et al. [62]. No units for the luminescence readings and calibration factors are given in [62]; the ɣ-doses are stated in rad in [62] but have here been converted to Gy.

is a valid question, but I will not pursue it and only note that Hashizume et al. [62] do not discuss it either. Instead, I will ask a simpler one: assuming that indeed the calibration factors may physically vary to such a large extent, how statistically likely is that they should do so in this very order, monotonously decreasing with increasing distance from the hypocenter? Since we have seven different values overall, that probability is $1/7! = 1/5040$, or roughly 0.0002.

The raw luminescence readings are substantially higher and more variable in samples from Nagasaki than in those from Hiroshima (Figure 4.4B).[8] Remarkably, however, the calibration factors vary exactly the opposite way, going up each time that L goes down, and down each time that L goes up, once more producing a smooth and regular curve for the ɣ-ray dose as a function of distance from the hypocenter. With again seven values overall and thus six transitions between them, the probability that they all would correspond this way by chance is $1/2^6$ or $1/64$. While this *is* almost a hundred times greater than the probability of the more regular trend in Hiroshima, it is still less than 5%, the threshold below which we conventionally reject random as a valid explanation. The *combined* probability that *both* data series should turn out the way they did is of course the product of the two numbers, or 3.1×10^{-6}.

[8] If there is any truth and relevance at all to the raw thermoluminescence readings, then the uniformly low values from Hiroshima may reflect widespread thermal inactivation due to the fire. Nagasaki was not as completely engulfed by fire, and thus more thermoluminescence activity—due to natural background, of course, not to any nuclear detonation—may have been preserved in those brick samples.

4.5 Conclusion

In this chapter, we examined two early and influential thermoluminescence studies that are still widely cited as evidence of ɣ-radiation from the detonations in Hiroshima and Nagasaki. We saw that in both studies essential precautions and controls are absent. Even more startlingly, in the one study that actually details at least some of its experimental results, the purported evidence of ɣ-radiation from the bomb is not apparent in the actual measurements of the bomb-induced thermoluminescence, but depends entirely on the stated results of the calibration procedure, whose falsity can be inferred from probabilistic arguments alone.

Since Hashizume et al. [62] obviously fabricated their evidence of ɣ-radiation, one may ask: why did they falsify the calibration factors rather than the readings of bomb-induced luminescence? The latter would have been more direct and also far more credible. I can't help thinking that they did it for this very reason—they *wanted* to be found out, to let the world know that their report was untruthful, while ostensibly conforming to the official lies and censorship imposed on them. Of course, this is just my own reading, which I cannot prove; readers will judge for themselves.

There is a number of more recent studies that use the same experimental approach, report largely similar findings, and are equally unconvincing with respect to sample selection. A fairly recent overview of the state of the 'art' can be found in Egbert and Kerr [65]; it lists two of the burnt-out buildings shown in Figure 4.3 as sampling sites. Remarkably, this latter paper also suggests that some thermoluminescence activation may have arisen not from direct irradiation during the detonation but later on from fallout—which, as we have seen in Chapter 2, carried only minuscule levels of radioactivity. Evidently, anything goes in this threadbare, disheveled field of 'science', as long as the conclusion toes the party line.

5. Statistical observations on acute 'radiation' sickness in Hiroshima and Nagasaki

> It is … difficult to explain the complete absence of radiation effects in … people who were theoretically exposed to lethal dosages of radiation.
>
> Ashley Oughterson and Shields Warren

In this chapter, we will examine the hypothesis that the victims in Hiroshima and Nagasaki suffered from *acute radiation sickness* (ARS), and in particular whether or not the pertinent statistical observations on this disease can be reconciled with current understanding in physics and medicine.

5.1 Physical assumptions

Before delving into the data themselves, we will note some assumptions which concern physical conditions and methods, and which will guide the interpretation of the medical data.

5.1.1 Radiation doses from fallout and induced radioactivity are negligible. As discussed earlier xref, the most important forms of radiation from a fission bomb are the ɣ-rays and neutron released during the blast itself. In contrast, residual radioactivity on the ground due to fallout and neutron capture should be minor; while it might pose some health risk in those exposed to it for long periods of time, it should not cause or contribute to acute radiation sickness. Cullings et al. [68] put it succinctly:

> *The radiation doses were truly acute, being received almost completely in a matter of seconds; furthermore, every person in each city received the dose at the same time … The situation regarding residual radiation was most recently reviewed in the DS86 Final Report.*[1] *As that report makes clear, doses from residual radiation are generally believed to be small …*

[1] See Roesch [69].

Note that the authors arrived at this conclusion when starting from orthodox tenets about the inner workings and the yields of the nuclear bombs. Thus, we don't need to assume that no nuclear detonations ever happened in order to dismiss fallout and neutron-induced radiation as possible causes of ARS; we are not making a circular argument.

5.1.2 Biology trumps physics in the detection of lethal radiation. All physical dosimeters and radiation counters are subject to measurement errors; but no frayed cable, leaky battery, or distracted operator can prevent the lethal effect of radiation on a human being.

The lethal dose of radiation for humans is approximately 8 Sv; with γ-radiation, this is the same as 8 Gy.[2] The only possible way to survive such a dose is through a bone marrow transplant, which of course was not available to the bombing victims. Indeed, total body irradiation with a lethal dose of γ-rays is one of two methods used to condition leukemia patients for a bone marrow transplant. Once a patient receives some 10 Gy of γ-rays as a single dose, his bone marrow *will* die—as will, hopefully, all of his leukemic cells, for that is the real purpose of the procedure; and so will *he*, unless transplanted with the bone marrow of a healthy donor immediately afterwards. Irradiation could not serve this purpose if it were anything but deadly every single time.

If a human being does not die, he or she did not receive a lethal dose; there can be no false-negative reading. Thus, if a physical measurement or estimate of radiation indicates that lethal radiation prevailed at a certain time and place, but a human being present then and there survived, then this biological outcome categorically falsifies the physical statement.

False-*positive* findings of sickness and death due to radiation can, of course, be produced with 'radiomimetic' compounds such as sulfur mustard; and accordingly the second conditioning method for bone marrow transplant is the use of drugs exactly of this kind.[3]

5.2 Manifestations of acute radiation sickness

The seriousness of acute radiation sickness depends, above all, on the dose of the radiation received. Other important considerations are whether that dose

[2] A benchmark that is easier to determine accurately than the 'always lethal dose' is the LD_{50}, that is, the dose that will be lethal to 50% of all individuals in a sufficiently large sample. The human LD_{50} has never been accurately determined; there simply are no adequate data. Under these circumstances, the best available substitute is the LD_{50} experimentally determined with rhesus monkeys (see Section 5.9).

[3] One early agent used for this purpose was in fact nitrogen mustard, which acts in exactly the same manner as does sulfur mustard. Nowadays, drugs are more commonly used than radiation.

is delivered all at once or in multiple sessions, and whether it is applied to the whole body or only to some body part. In a nuclear detonation, irradiation will usually affect the whole body evenly, and all doses stated in the following should accordingly be taken as whole-body doses.[4] Also important are type and particle energy of the radiation; this is discussed in Section A.9.2.

The sensitivity to radiation differs greatly between tissues and cell types in the body, and therefore different organs will respond at different threshold doses. Three sub-syndromes that concern different target organs can be distinguished.

5.2.1 The hematopoetic syndrome. This syndrome is caused by damage to bone marrow stem cells, which are among the most radiosensitive cell types.[5] It becomes manifest at doses above 1.5-2 Sv, and no patients who received more 5-6 Sv will survive if intensive medical care is unavailable. All types of blood cells are descended from bone marrow stem cells, and thus all of them will fail to be renewed in hematopoetic syndrome (HS for short). However, the consequences are most dramatic with the white blood cells and with thrombocytes, since these are short-lived (see Figure 5.4). In contrast, mature red blood cells have a life span of 120 days; they can sustain the patient even when their regeneration ceases for several weeks, and they thus will not limit his lifespan in the acute phase of HS.

When leukocytes fail, the patients will suffer from infections; when platelets are depleted, bleeding will occur spontaneously or after minor trauma. Numerous scattered hemorrhagic spots will arise that are most readily observed beneath the skin or the mucous membranes of the oral cavity, but which equally affect the inner organs; and in severe cases, the patient may bleed to death internally. This condition is referred to as *purpura*, and the characteristic hemorrhagic spots are called *petechiae*.

As long as some bone marrow stem cells survive, blood cell formation will eventually resume; if levels of white blood cells and of platelets fall dangerously low, they may be transiently substituted by transfusion. If all stem cells were wiped out, then only a transplant of bone marrow from a compatible donor can possibly save the patient.

Radiation doses similar to those that damage the bone marrow will also damage the hair follicles. In this case, too, loss of function may be transient or

[4] Local cancer radiotherapy often uses very high doses that would be lethal if applied to the whole body.

[5] The cells of the bone marrow are shielded to some degree from natural radiation by the mineral of the surrounding bone matrix. Did natural selection hide them there because they were sensitive, or did they evolve to be sensitive because they were shielded?

permanent; higher doses will cause greater loss of hair, and permanent hair loss will occur at doses similar to those that irreversibly destroy the bone marrow. Thus, hair loss provides a useful proxy for estimating the extent of damage to the bone marrow.

5.2.2 The gastrointestinal syndrome. At doses of 6 Sv and above, damage to the intestines will give rise to diarrhea and often outright intestinal bleeding. The breakdown of the gut barrier will facilitate infections, which will be made worse by the depletion of white blood cells. Loss of fluid and electrolytes will further aggravate the situation. Intensive care with antibiotics and replacement of fluids and electrolytes, in addition to treatment of the hematopoetic syndrome, may rescue patients with doses up to 10 Sv, but at dosages higher than this the prognosis of gastrointestinal syndrome becomes hopeless. Of course, none of these therapeutic measures were available in Hiroshima and Nagasaki; under those conditions, practically all patients with manifest gastrointestinal syndrome should have died.

5.2.3 The cerebrovascular syndrome. At very high doses—the threshold doses given in the literature vary considerably, reflecting the paucity of clearly documented cases; but a widely cited IAEA report states 20 Gy [70]—radiation will kill within 1-2 days by direct action on the central nervous system. It is believed that damage primarily affects the small blood vessels in the brain; inhibited perfusion then causes various manifestations of brain dysfunction.

Hall and Giaccia [71, p. 218] point out that, even though neurological symptoms may dominate the clinical picture, the damage to the vascular system is likely general. This matches their case descriptions of two workers who developed cerebrovascular syndrome after receiving extremely high doses of irradiation by accident, and who also suffered general circulatory shock, to which they succumbed within two days after the exposure.

5.2.4 Prodromal and latent stages. All three syndromes described above take days or weeks to become fully manifest; and, for reasons explained in Section A.11, the delay will be longer with lower radiation doses. Minutes to hours after exposure, however, there will be some early signs, less severe and less characteristic. Most common at this *prodromal* stage are vomiting and mild headache; diarrhea and fever indicate higher doses and presage later manifestation of gastrointestinal syndrome. In all but the most severe cases, these prodromal signs subside, and the patients will enter a *latent* stage showing few clinical symptoms or none at all. During this time, however, cell proliferation within the bone marrow and, at higher doses, within the intestine drops off,

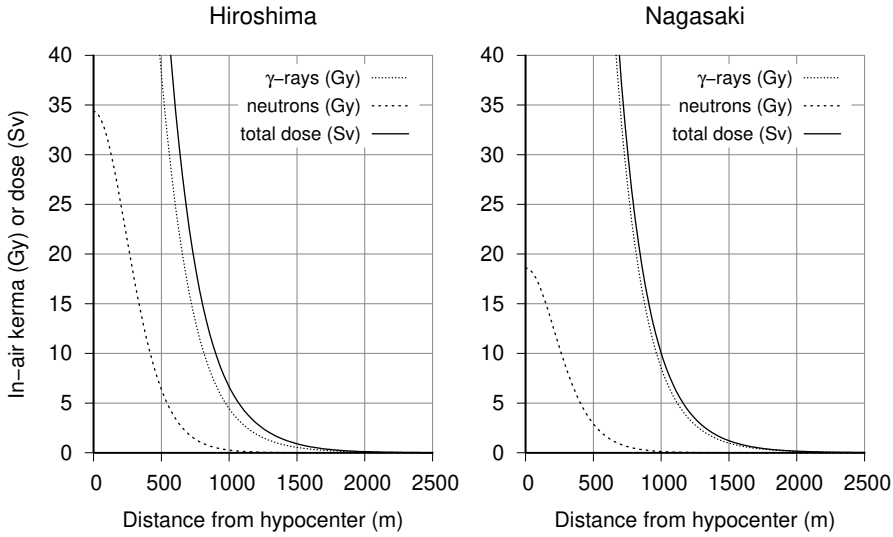


Figure 5.1 Estimated radiation dosages at Hiroshima and Nagasaki, as a function of distance from the hypocenter. The in-air kerma for γ-rays and neutrons was taken from Cullings et al. [68]. To calculate the total dose, the dose-dependent relative biological effect of neutrons was estimated according to Sasaki et al. [72] (see text for details).

and the specific syndromes manifest themselves once the initially surviving maturing or fully mature cells in these organs expire.

5.3 Acute radiation doses in Hiroshima and Nagasaki

The tenet that, in Hiroshima and Nagasaki, doses sufficient to cause acute radiation sickness could have been inflicted only during the blast itself (see Section 5.1.1) gives rise to a number of testable predictions, which we will examine in the following.

5.3.1 Radiation dose as a function of distance from the hypocenter. Since there were no instruments in place to measure the radiation doses when the detonations occurred, we have to make do with approximations based on indirect methods and calculations. The officially endorsed dose estimates have seen some fairly considerable changes over time. Figure 5.1 depicts the biologically effective or equivalent doses for both Hiroshima and Nagasaki, based on current estimates of γ-ray and neutron intensities [68]. In this graph, the biological dose was calculated by applying an experimentally determined dose-dependent relative biological efficiency (RBE) function for neutron radiation [72] to the neutron component of Cullings' neutron radiation levels.

5.3.2 Shielding from radiation by buildings. The dose estimates in Figure 5.1 apply to persons who were directly in the path of the radiation, without any sort of solid matter between them and the site of the detonation up in the air (the epicenter). However, many people were indoors at the time of the bombing, and some of those who found themselves outdoors were shaded from the detonation by some intervening structure.

Traditional Japanese houses were simple buildings with one or two stories, constructed mainly from wood, sometimes with thatched roofs but mostly with tiled ones. This was the predominant type of building in both Hiroshima and in Nagasaki, although in the latter city the proportion of concrete buildings is said to have been somewhat higher. The penetration of γ-rays and fast neutrons into such traditionally constructed buildings was studied quite thoroughly in the 1950s and 60s, as documented by Auxier [33] and Arakawa [73]. According to these measurements, γ-ray doses inside such buildings would have been ≥ 60%, and neutron doses ≥ 40% of those in the open. Thus, these buildings would have given only very limited protection from bomb radiation. In contrast, buildings constructed from concrete could have provided effective shielding, particularly within rooms facing away from the detonation.

5.3.3 Threshold distances for radiation doses. Considering the almost complete lack of medical care available to the bombing victims, we can assume that survival of more than 6 Sv would have been impossible; according to the estimate shown in Figure 5.1, this threshold is reached or exceeded in both cities at distances up to 1000 m. Accordingly, there should have been no possibility of surviving an unshielded exposure within 1000 m in either Hiroshima or Nagasaki. Within 500 m, unshielded doses should invariably have caused cerebrovascular syndrome, the most severe and rapidly deadly form of ARS; and this should apply not only to persons without shielding, but also to those shielded by no more than a traditional wooden house. On the other hand, beyond 1500 m in both cities, the unshielded dose drops to a level below which no serious manifestations of acute radiation sickness are to be expected.

5.3.4 Predicted distance distribution of ARS. From the foregoing observations, we can conclude that the statistics on ARS in Hiroshima and Nagasaki should exhibit a highly regular pattern, with the following characteristics:

1. within 500 m, all of those exposed without shielding or inside traditional wooden houses should have suffered cerebrovascular syndrome, and none of them should have survived beyond 2-3 days;

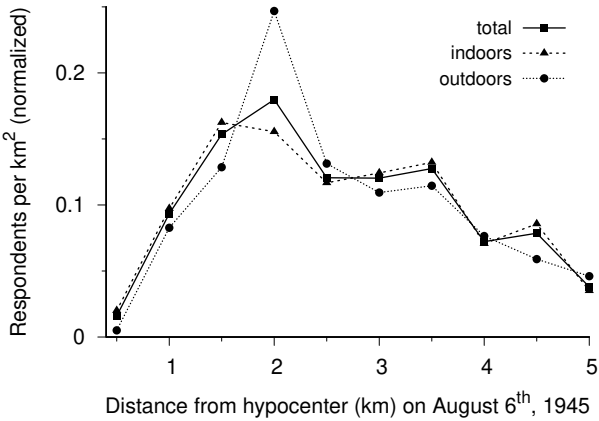


Figure 5.2 Distribution of survivors in Hiroshima, by shielding (indoors/outdoors) and distance from the hypocenter on the day of the bombing. A survey in 1957 [29] canvassed all persons then living within 7 km of the hypocenter. The area near the hypocenter shows a very low number of respondents per km^2, which likely reflects survival rates; the decrease above 2 km may simply be due to lower population density in the suburbs. Data from Tables 1-4 in [29].

2. between 0.5 and 1 km, ARS should have occurred in all persons exposed inside wooden houses or without shielding; and in the latter group, there should be no survivors;
3. between 1 km and 1.5 km, a very large proportion of victims who were exposed with light shielding or in the open should have suffered ARS, ranging from mild and transient to violent and deadly;
4. at most a few, light cases of ARS should have occurred among those exposed beyond 1.5 km, regardless of shielding;
5. absolutely no ARS cases whatsoever should have occurred beyond 2 km.

Note that these threshold distance values are based on current dose estimates. Early estimates were substantially higher (see for example Arakawa [73]), and the publicly available literature really doesn't offer any substantial evidence that would support the currently preferred lower ones. If we assume that those earlier numbers were in fact correct, then a similar pattern should still emerge, but with each of the boundaries stated in the list above approximately 500 m further out.

5.4 Observed distance distribution of ARS in Hiroshima

We will now compare observed occurrences of ARS and of survival to predicted ones. The two key sources for this purpose are Oughterson et al. [28] and Sutou

[29]. Both studies report statistics on several thousand individuals. The first one was compiled by the 'Joint Commission for the Investigation of the Effects of the Atomic Bomb in Japan', a group of American and Japanese physicians convened at the initiative of Ashley W. Oughterson, a professor of surgery who at the time was serving as a colonel in the U.S. military. This commission only arrived at Hiroshima and Nagasaki in October 1945, but it did acquire and organize data previously collected by Japanese physicians; and the statistical evaluation of these earlier Japanese data forms indeed the main substance of the commission's report. Most of the figures tabulated in [28] pertain to patients still alive and in medical care at 20 days after the bombings;[6] recorded are slightly below 7000 survivors in each city.

The second study was carried out in 1957 by Dr. Gensaku Oho,[7] a physician from Hiroshima, who enlisted the help of student volunteers to canvas the resident population of Hiroshima. The main purpose of this study was to determine the occurrence of radiation sickness among persons who had not been exposed to the bombings themselves, but who had entered the area close to the hypocenter only afterwards. The more recent paper by Sutou [29] which is used here is a partial translation of and commentary on this earlier study.

5.4.1 Survival of persons exposed within 500 m of the hypocenter. The first prediction is that no one should have survived beyond a few days who was exposed, with light shielding or without it, within 500 m of the hypocenter. This prediction is falsified by the following findings:

1. Twelve of Oho's respondents in 1957 reported having been exposed within 0.5 km of the hypocenter. Of these, one had been exposed outdoors, whereas eleven had been indoors; presumably, at least some among this number had been inside wooden buildings.
2. Keller [9] lists eight patients at Osaka University Hospital as having been exposed inside wooden buildings within no more than 500 m, and among them four had been within 50 m. He further states that of all patients in his survey five succumbed, and that the average day of death among these five was 26 days after the bombing. Therefore, at least three patients exposed within 500 m the hypocenter were still alive some four weeks after the bombing. Even the patients who did succumb within four weeks had

[6] For an account of the pitiful conditions these patients were suffering at the time, see for example the book by Swiss ICRC physician Junod [31], as well as the short film *Hiroshima-Nagasaki 1945* [74].

[7] The last name is transcribed as 'Obo' by [29] and as 'O-ho' in some other sources. Not knowing which spelling is the most appropriate, I adopted the one which I saw used most widely.

Table 5.1 Prevalence of specific symptoms of acute radiation sickness—epilation and/or purpura (E/P)—among patients in Hiroshima who were still alive 20 days after the bombing, by distance from hypocenter and type of shielding. The columns labeled with † give the numbers of patients known to have died later. 'Japanese' buildings are understood to be of traditional, wooden construction. Excerpted from Tables 59H and 68H in Oughterson et al. [28].

	Outdoors, unshielded			Inside Japanese building		
Distance (km)	Alive at 20 d	E/P (%)	†	Alive at 20 d	E/P (%)	†
0–1.0	105	88.6	22	410	85.9	120
1.1–1.5	249	42.6	9	560	38.6	19
1.6–2.0	689	14.2	4	754	10.1	3
2.1–2.5	590	6.8	1	731	4.7	0
2.6–3.0	192	7.8	0	390	2.6	0
3.1–4.0	159	3.8	0	325	1.2	0
4.1–5.0	68	2.9	0	127	0.8	0

survived long enough to be transported to Osaka, and therefore must have lived longer than compatible with cerebrovascular syndrome.

This number of confirmed survivors is certainly very small, which means that the inferno in the city center must have been every bit as deadly as eyewitness testimony indicates [75, 76] (see also Figure 5.2). Nevertheless, if we accept that there are any survivors at all, *then this finding alone disproves the story of the nuclear detonation, and no amount of physical studies can possibly salvage it*—remember that no false-negative measurements are possible with our *Homo sapiens* reference dosimeter.

5.4.2 Survival and incidence of ARS among patients exposed within 1 km of the hypocenter. Oughterson et al. [28] do not separate exposure within 0.5 km from that within 1 km, presumably because they considered the numbers in the former group too low. However, beginning with 1 km, they group patients by distance intervals of 0.5 km, and they carefully subdivide each group according to different types of shielding. Table 5.1 contains a selection of these data, on which we can make the following observations:

1. On the twentieth day, 88.6% of patients exposed within 1 km and in the open have developed specific symptoms of radiation sickness, which means that 11.4% have not. Similar proportions are found with those who were exposed while inside Japanese style houses.

With doses as high as the those predicted for this range, the latency period of ARS should last at most 8-18 days [26]. Therefore, the observation of patients who on the 20th day still show no signs of manifest ARS deviates from expectation.

2. Of the 105 patients exposed in the open and still alive on the 20th day, only 22 are known to have died later on. Oughterson et al. [28] quite sensibly state that

   it is probable that other unreported deaths occurred in this group of people, and some may have died as a result of radiation after the end of the survey in Japan.

   However, they also show (in their Table 58) that death rates steadily declined as time went on. Out of a total of 6663 patients recorded in Hiroshima as being alive on the 20th day, 254 or 4% are reported to have died subsequently. 137 of these deaths occurred between days 20 and 29, whereas only two occurred between days 70 and 79, and another five occurred between day 80 and the unspecified end date of the survey. Considering this time course, it is highly likely that most of the 83 patients who had been exposed in the open within 1 km, and who had survived the entire time period of the study, also remained alive thereafter—in marked contrast to the expectation that they should all have perished.

In summary, while the proportion of ARS sufferers in this group is large, it is not quite as large as predicted, and the number of long-term survivors deviates from prediction even more clearly.

5.4.3 Incidence of ARS at >1 km from the hypocenter. Above, we stated that a large proportion of persons within 1-1.5 km should suffer from ARS. In patients exposed without shielding or with light shielding only, the proportion listed in Table 5.1 is close to 40%. While this is low, we must allow that in some cases the symptoms may not yet have been manifest on the survey's reference date, for at dosages below 4 Gy the latency period may exceed 20 days [26]. In contrast, the mortality is again implausibly low. The ARS cases observed beyond 2 km from the hypocenter—at frequencies below 10% and decreasing with distance, but not quite dropping to zero even between 4 and 5 km—differ from expectation unequivocally; they are not explained even by the highest published estimates of acute radiation doses.

The above findings were confirmed by Oho, who documented cases ARS among survivors that had been at ≥ 2 and even ≥ 3 km from the hypocenter

during the detonation. Importantly, this applied even to some survivors who had stayed away from the hypocenter for several weeks after the bombing [29].

5.5 Observed distance distribution of ARS in Nagasaki

The observations made above for Hiroshima mostly apply to Nagasaki as well (see Table 68N in [28]); however, some findings are quantitatively more pronounced. ARS symptoms and mortality are less frequent within 1 km than in Hiroshima, even though radiation doses are supposed to have been higher (Figure 5.1): among survivors exposed in the open or shielded only by a wooden house, less than 60% exhibit epilation or purpura. Among survivors exposed between 1.5-2.5 km, a greater percentage than at Hiroshima shows symptoms of ARS. On the other hand, beyond 4 km from the hypocenter, that percentage does indeed drop to zero in Nagasaki, whereas it stays slightly positive at this distance in Hiroshima.

5.6 ARS symptoms in people shielded by concrete buildings

Concrete buildings will afford substantial protection from both ɣ-rays and neutron radiation, and we should therefore expect a lower number of ARS victims among those inside these buildings than in those inside wooden buildings or in the open. This is indeed observed; within 1 km from the hypocenter, the incidence of ARS is approximately 25% lower inside heavy buildings than outside, both in Hiroshima and Nagasaki ([28], Tables 68H and 68N). Yet, ARS inside heavy buildings in Hiroshima remains more abundant than it is in the open in Nagasaki, even though the radiation dose is said to have been higher in Nagasaki.[8]

More detailed statistics on this question are reported by Oughterson and Warren [78], who in their Table 3.7 show findings from three individual concrete buildings in Hiroshima, all of which were situated between 700 and 900 meters from the hypocenter. In each building, some people were protected by multiple walls or floors, such that the total shielding was equivalent to ≥ 154 inches (or 394 cm) of water (see Table 5.2). The stated radiation dose *outside* the buildings was up to 80 Gy, which amounts to approximately ten times the lethal dose. However, after passing through this much shielding, it should have been attenuated to a mere 4 mGy. This corresponds to just 2/3 of the typical annual dose of a U.S. citizen and will, of course, not produce any acute symptoms at all.

Nevertheless, Oughterson and Warren [78] report cases of ARS—some of them lethal—in persons thus protected. They propose that these may be due

[8] Possible reasons why concrete buildings would have offered partial protection from mustard gas will be considered elsewhere xref.

Table 5.2 Attenuation of ɣ-rays and fast neutrons by different materials. Numbers are estimates of the layer thickness that would have reduced initial ɣ-ray dosage in Hiroshima by 90%. Data for ɣ-rays from Ishikawa et al. [7] (p. 72); value for neutrons and concrete calculated from numbers given in Yılmaz et al. [77].

	Layer effecting 90% attenuation (cm)	
Material	ɣ-rays	fast neutrons
Iron	9-13	
Concrete	30-45	13
Wood	125-175	
Water	65-92	
Soil	45-65	

to neutrons, apparently assuming that neutrons are less effectively shielded by concrete than are ɣ-rays. However, this is now known to be incorrect (see Table 5.2); and moreover, as already noted, the estimated neutron dose at Hiroshima was very substantially reduced in the decades after their book was published [79].

As a second *deus ex machina*, the authors suggest that the bomb's ɣ-radiation may have been of much higher particle energy, and therefore more penetrating, than is generally assumed. However, they do not offer a physical basis for this hypothesis, nor do they pursue its wider implications for the physical and medical dosimetry of the entire event, which would have been substantial. Such lack of interest suggests that the authors themselves do not take their own proposal seriously. When commenting on the reverse scenario—the wondrous survival of some individuals exposed to strong ɣ-radiation—the authors dispense with any special pleading and blankly state (p. 63):

> *It is equally difficult to explain the complete absence of radiation effects in a number of people who were theoretically exposed to lethal dosages of radiation.*

We note that Oughterson and Warren [78] acknowledge the dual quandary of ARS occurring among those beyond the reach of the bomb's radiation, while failing to appear in some of those exposed to a 'theoretically lethal' dose. Adjusting dose estimates will not solve this dilemma: increasing doses may avoid the Scylla of death despite protection, but it will wreck the ship on the Charybdis of inexplicable survival; reducing doses to explain miraculous survival renders the deaths of shielded victims all the more incomprehensible.

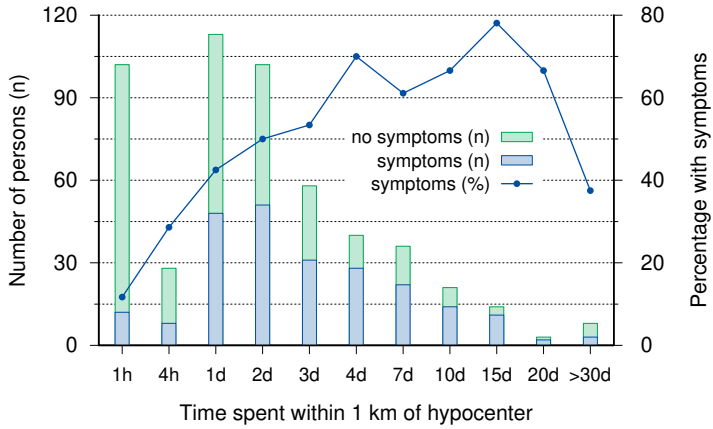


Figure 5.3 Symptoms of ARS in persons who were outside Hiroshima during the bombing, but came within 1 km of the hypocenter afterwards, as a function of time spent in that area. Data from Table 7 in [29]. Symptoms of ARS include fever, diarrhea, bloody stools, bleeding from the mucous membranes, loss of hair, and generalized weakness.

5.7 Symptoms of ARS in people who entered central Hiroshima after the bombing

The occurrence of ARS symptoms in persons who were outside Hiroshima on the day of the bombing but entered the zone within 1 km of the hypocenter afterwards is a crucial piece of evidence. While anecdotal reports are found in many sources, e.g. [12, 27, 47, 75], there is only one statistical study on this question; and it is telling that we owe this study to the personal initiative of an energetic doctor from Hiroshima, Gensaku Oho, and his student volunteers, rather than to the official institutions created and maintained for such investigations by the governments of the United States and of Japan.

A summary of Oho's most important findings is given in Figure 5.3. Many people entering the area within 1 km of the hypocenter[9] report symptoms of ARS; the percentage of people thus affected exceeds 50% among those who stayed for more than 2 days. Additional tables and figures presented by Sutou [29] clearly document that the same effect is also present among those who were in Hiroshima during the bombing: while of course many in this group suffered ARS regardless of their whereabouts in the aftermath, the incidence is higher among those who also came near the hypocenter in that period.

[9]The text in reference [29] states distances from the 'epicenter'; however, in direct correspondence, the author confirmed that the intended meaning is 'the ground site right under the detonation', for which the term 'hypocenter' is conventionally used.

Findings such as those reported by Oho can, of course, not be explained with the radiation released during the detonation. There are three ways of dealing with this problem:

1. The findings are ascribed to fallout or residual radiation, which are assumed to have been much greater than official estimates [29, 80, 81].
2. The findings are declared to 'warrant further analysis' and then studiously ignored [33, p. 90].
3. The findings are ignored without ceremony. In case you guessed that this is the most common approach, you are indeed correct.

The last two alternatives require no comment. Regarding the first one, it was shown earlier that real fallout must have been lower, not higher than the official estimates, and there is no basis whatsoever for higher estimates of neutron-induced radioactivity.[10]

The thesis of this book—namely, that sulfur mustard, not radiation was the cause of 'ARS'—provides a ready explanation for cases of the disease among late entrants to the city. Sulfur mustard is known to linger, and its persistent stench was noted by Burchett four weeks after the bombing [12]. Wind-driven mustard fumes would explain why those located downwind from the hypocenter suffered more 'ARS' [80] and were at greater risk of developing cancer [82, 83]. While Yamada and Jones [80] ascribe the surplus incidence of ARS in this group to high β-radiation from isotopes contained in the black rain, the very low levels of ^{137}Cs in extant black rain samples [5] disprove their explanation.[11]

5.8 Late onset ARS

In patients who suffered ARS due to irradiation or poisoning only after the bombing, the symptoms should develop with some delay; and this is indeed reflected in the statistics reported by Oughterson et al. [28].

[10] It is remarkable how two mutually exclusive narratives—harmful radiation released in the blast only, and major contribution from fallout or induced radiation—have co-existed peacefully for many decades in the literature. In this field of 'research', hard questions are never answered, but deferred and dodged forever—if need be, as in this case, through the use of Orwellian doublethink.

[11] The wind is said to have blown toward the west at Hiroshima [82]. Yamada and Jones [80] do not specify where in the city their black rain victims had been located. However, Masuda in [84] gives a detailed map, constructed from statements obtained from many survivors, which indicates that the black rain was most intense in the northwest.

While Peterson et al. [82] find cancer incidence increased in the west, Gilbert and Ohara [85] find acute radiation disease most abundant in the north, but below average in the west. ARS requires high doses, whereas cancer may be caused in a large enough population by lower doses also; therefore, the observed discrepancy suggests a fairly uneven distribution of mustard gas.

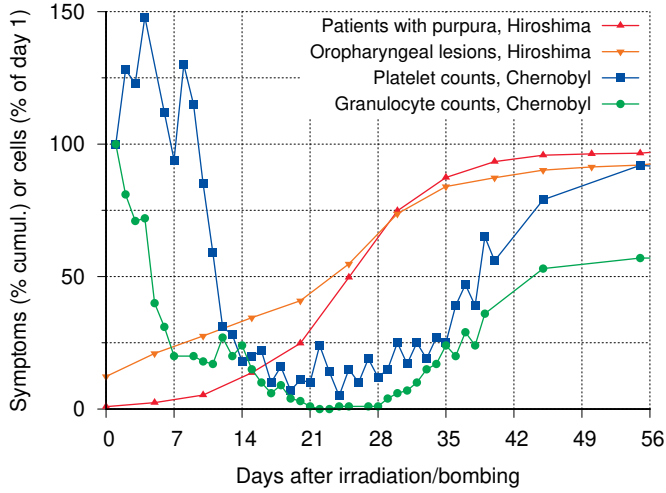


Figure 5.4 Time of onset of purpura and oropharyngeal lesions in Hiroshima bombing victims, and blood cell counts in accidentally irradiated patients. Data for onset of purpura (bleeding) and oropharyngeal lesions in Hiroshima victims from Oughterson et al. [28, Table 17H]; 100% is the total of all patients that exhibited the symptom at any time during the observation period. Platelet and granulocyte counts (from Fliedner et al. [86]) represent median values of 11 patients who were exposed to whole body irradiation at Chernobyl. Values are relative to those on day 1, which were in the normal range for both cell types.

Characteristic symptoms of ARS hematopoetic syndrome (see Section 5.2.1) are purpura, caused by the failure of the blood platelets, and oropharyngeal ulcers due to bacterial and fungal infections, which are brought on by the lack of granulocytes. In patients who exhibit these symptoms after exposure to a single dose of irradiation, they become manifest between days 8 and 28, with shorter latency at higher doses [70]. Figure 5.4 shows that this is also true of most Hiroshima bombing victims; however, in about 25%, the initial manifestation is delayed until the fifth week or later.[12] For illustration, the figure also shows the time course of platelet and granulocyte counts in patients exposed to irradiation at Chernobyl. Both cell counts reach their lowest point before the 28$^{\text{th}}$ day, which explains that symptoms will be manifest by this time.

[12]It is also interesting to note that oropharyngeal lesions are manifest in a considerable number of Hiroshima bombing victims in within the first week, and even on the first day. It seems likely that these very early lesions are due to direct, local action of inhaled or ingested sulfur mustard rather than to hematopoetic syndrome.

Anecdotal evidence confirms the occurrence of late cases. For example, in his posthumously[13] published book *First into Nagasaki* [88], the American journalist George Weller notes on September 22nd:

> *New cases of atomic bomb poisoning with an approximate fifty percent death rate are still appearing at Nagasaki's hospital six weeks after the blow fell, but United States Navy physicians who have examined them report that the death rate is falling off.*

The decreasing, yet still ongoing observation of new cases agrees with the data in Figure 5.4. While from this limited information we cannot be sure whether the death rate in new cases was indeed falling, this would be plausible in real ARS [70] and similarly also in mustard poisoning. What is *not* plausible in true ARS , however, is the repeated occurrence of new cases, particularly ones with fatal outcome, as late as six weeks after the exposure. These patients must have taken in the poison some after the bombings, just like some of the subjects surveyed by Oho [29].[14]

5.9 ARS symptoms and official radiation dose estimates

We have seen earlier that radiation measurements at the time of the bombings or in the immediate aftermath had been very limited in scope, and accordingly early medical studies could only guess at the radiation doses received by individual survivors (see for example Wilson [55] and Lindee [37, p. 197ff]). When the 'Life Span Study'—a large-scale program of medical surveillance among the survivors, which is still ongoing— was begun in the late 1950s, an attempt was also made to address this lack of information on individual radiation exposure. As described in detail by Auxier [33], various large-scale physical experiments were carried out, often in conjunction with ongoing nuclear bomb tests, in order to determine the in-air doses of ɣ- and neutron radiation that would have prevailed at various distances from the hypocenters in Hiroshima and Nagasaki. From these values,

[13] Both Hiroshima and Nagasaki had been declared out of bounds for civilians by MacArthur, but just like Burchett sneaked into Hiroshima [12, 87], so Weller stole into Nagasaki. Unlike Burchett, however, Weller still dutifully filed his reports with McArthur's censors, who prohibited their publication. Weller did retain a copy, which was found in his estate by his son, who edited and finally published it in 2007.

[14] Poison in the air was noticeable for several weeks after the bombings also at Nagasaki. Tatsuichiro Akizuki, a Nagasaki physician, vividly describes how a heavy rainstorm pelted yet cleansed the city on September 2nd and 3rd [89, p. 135]: "I looked up at the sky and shouted: 'Don't punish them this way—it is too much! Haven't you done enough?' ... The 4 September turned out to be a fine, cool, autumn day. ... 'Something has happened!' I said to Miss Murai. 'I feel there's a change in the air—I'm sure of it.' ... 'That's it!' I said to myself. The poison has been washed away!"

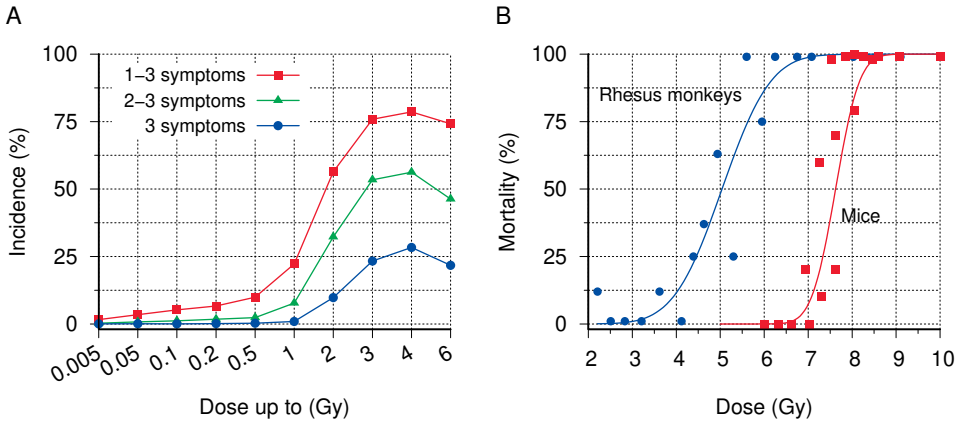


Figure 5.5 Incidence of ARS symptoms and radiation dose estimates in atomic bomb survivors, and experimental mortality curves in mice and rhesus monkeys. A: Incidence of ARS symptoms vs. radiation dose groups. The three symptoms of ARS reported in the data set [90] are epilation, bleeding, and oropharyngeal lesions. B: Mortality in rhesus monkeys exposed during a series of bomb tests [91] and in mice subjected to single doses of 250 kV X-rays [92]. Curves are fits to a cumulative Gaussian distribution.

the individual radiation doses were then derived by taking into account the specifics of location and shielding for each survivor, as gathered from detailed interviews. The initial data set thus obtained, released in 1965, became known as 'Tentative 65 Doses' (T65D) and was used until 1986, when it was replaced with the 'Dosimetry Scheme 1986' (DS86) xref.

While these dose estimates are widely accepted and used as reference values in many studies on radiation effects, a comparison with the incidence of ARS symptoms reveals the same flaws and discrepancies already noted in the preceding sections. Figure 5.5 A shows the incidence of three ARS symptoms—epilation, bleeding, and oropharyngeal lesions, alone or in combination—as a function of estimated radiation dose. Up to 0.5-1 Gy, none of these symptoms should be present, but they clearly do show up at these low estimated doses. On the other hand, above 3-4 Gy essentially all patients should have experienced at least two of the three symptoms.[15] Nevertheless, even at these very high doses, the fraction of patients with two or more symptoms does not exceed 60%. Also peculiar is the drop in symptom incidence in the highest dose group.

We can compare the ARS symptom trends to proper dose-response curves observed in rhesus monkeys exposed to mixed ɣ- and neutron radiation dur-

[15] While oropharyngeal lesions involve infection and might be averted by rigorous hygiene—which of course was not feasible under the conditions prevailing after the bombings—bleeding and epilation would by unavoidable even with optimal care.

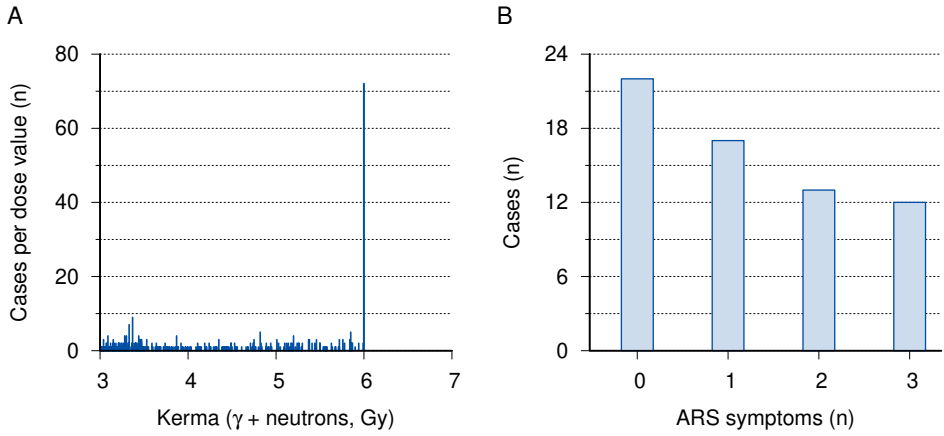


Figure 5.6 Numbers of survivors grouped by dose values (A), and incidence of ARS symptoms among those assigned an estimated dose of 6 Gy (B). In RERF's dataset [90], one or more symptoms were given as 'not reported' for 8 out of the 72 survivors with exactly 6.000 Gy; these are included in A but excluded in B.

ing a series of bomb tests, as well as in mice experimentally exposed to X-rays (Figure 5.5 B). While the mice can tolerate higher radiation doses than the monkeys, both curves exhibit a very steep transition from very low levels of response—in this case, mortality—to very high ones; doses that cause almost complete mortality exceed those which cause virtually none by a factor of no more than 2. The results obtained with rhesus monkeys, in particular, are more than merely an illustration, since these monkeys are physiologically very similar to humans and thus provide the best animal model for estimating human radiosensitivity.[16] If a dose of 5 Gy is sufficient to kill 50% of the rhesus monkeys, and one above 6 Gy kill almost all of them, it simply is wildly implausible that fully 25% of humans exposed to the same dose would show not even one major symptom of acute radiation sickness, and fully 75% no more than two.

Figure 5.5 A used the low-resolution dose groups predefined by the data set, which was obtained from RERF.[17] In addition, the data set also contains

[16]Primates, including both humans and rhesus monkeys, share some metabolic traits likely to affect susceptibility to radiation. They require ascorbic acid as a vitamin, while also degrading adenine and guanine to uric acid. Radiation effects are mediated by radicals (Section A.11); both ascorbic acid (vitamin C) and uric acid can scavenge radicals and thus mitigate radiation effects.

[17]RERF stipulates that each work that includes any of their data contain the following statement: "This report makes use of data obtained from the Radiation Effects Research Foundation (RERF) in Hiroshima, Japan. RERF is a private foundation funded equally by the Japanese Ministry of Health, Labour and Welfare and the U.S. Department of Energy through the U.S. National Academy of Sciences." Furthermore, I am to say that "the conclusions in this report [the one you are reading]

radiation doses at higher resolution. If we plot a histogram of the number of people grouped by the individual dose values in the file, we see that the dataset contains no cases with estimated doses above 6 Gy (Figure 5.6 A). However, the number of people with an assigned dose of exactly 6.000 Gy greatly exceeds that of any other individual dose value above 3 Gy; in fact, only below 1 Gy do we find dose values with higher head counts than 6 Gy exactly. This peculiar pattern strongly suggests that all raw dose estimates higher than 6 Gy were simply truncated at that value; probably because they were deemed unsurvivable, and quite possibly under the impression of the rhesus monkey experiments shown in Figure 5.5. It should go without saying that such sausage-making does not qualify as science. Furthermore, whether truncated or not, in this highest of all dose groups, the number of individuals with 0 or only one symptom of ARS exceeds that with two or more symptoms (Figure 5.6 B). The 22 individuals without any symptoms clearly count among Shields' and Oughterson's mystery patients with 'complete absence of radiation effects' in spite of exposure to 'theoretically lethal' doses of radiation.

The findings in this section reinforce our previous observation that the distribution of ARS does not fit the official story of the bomb and its radiation. What is more, they clearly show that RERF's official radiation dose estimates correlate very poorly with their own records of ARS. *RERF must therefore be aware that their dose estimates are completely inadequate*, and their continuing use in statistical studies on the health of survivors is deceptive and indefensible. We will revisit the question of purported radiation doses and biological effects in Chapter **??**.

5.10 Diarrhea as an early symptom of ARS

Before leaving this topic, one recurrent motif in the reports on 'radiation sickness' from Hiroshima and Nagasaki should be noted: the widespread and early occurrence of diarrhea, often bloody, among the patients. A graphic account is given by Michihiko Hachiya [47]. The author, a head physician who had been injured in th bombing and admitted as a patient to his own hospital, wrote in his diary on August 7th:

> *Everything was in disorder. And to make matters worse was the vomiting and diarrhea. Patients who could not walk urinated and defecated where they lay. Those who could walk would feel their way to the exits and relieve themselves there. Persons entering or leaving the hospital could*

are those of the authors and do not necessarily reflect the scientific judgment of RERF or its funding agencies." To which I add: I bet they don't.

not avoid stepping in the filth, so closely was it spread. The front entrance became covered with feces overnight, and nothing could be done for there were no bed pans and, even if there had been, no one to carry them to the patients.

Disposing of the dead was a minor problem, but to clean the rooms and corridors of urine, feces, and vomitus was impossible.

Such events would suggest an outbreak of some virulent enteric pathogen, which is indeed common in disaster situations; and Hachiya and his staff initially assumed this to be the case. On August 7th, Hachiya writes:

Dr. Hanaoka ... brought word that there were many who not only had diarrhea but bloody stools and that some had had as many as forty to fifty stools during the previous night.[18] *This convinced me that we were dealing with bacillary dysentery and had no choice but to isolate those who were infected.*

Dr. Koyama, as deputy director, was given the responsibility of setting up an isolation ward.

However, already on August 13th, he notes:

My conjecture that deaths were due to the effects of a germ bomb causing dysentery I had to discard because diarrhea and bloody stools were decreasing.

Hachiya's conclusions are confirmed by the data given in Oughterson et al. [28], which show that both bloody and non-bloody diarrhea are strongly correlated with other ARS symptoms, and also that case numbers were highest early on and then declined, even though the hygienic conditions remained about as bad as can be imagined.

Diarrhea can indeed occur in real radiation sickness. However, it commonly occurs very early on only in patients who have received a dose of 6 Sv or greater [70]. Under the conditions then prevailing in Hiroshima and Nagasaki, patients hit with such a high dose would not have survived. Yet, the data listed by Oughterson et al. [28] pertain to patients who were alive 20 days after the bombing, and 96% of whom remained alive when the study concluded several months later (see Section 5.4.2), which means that they were not lethally irradiated. Thus, the timing of diarrhea observed in Hiroshima also indicates that the 'ARS' was not actually caused by radiation. On the other hand, early onset diarrhea has

[18] These cases are unlikely to have survived, and they will therefore be missing from Oughterson's statistics.

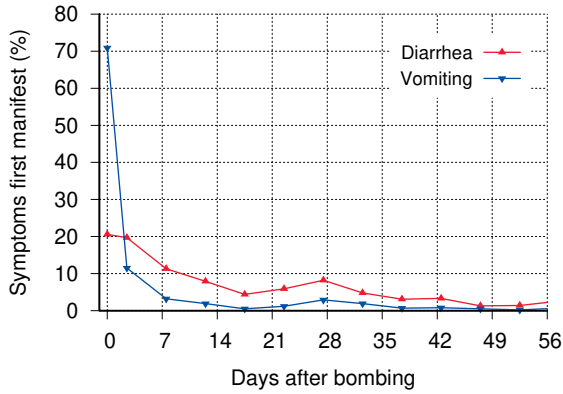


Figure 5.7 Time of onset of diarrhea and vomiting in Hiroshima bombing victims. Data from Table 18H in [28]. The first data point in each series represents the day of the bombing.

been described in multiple reports on mustard gas exposure of humans and of experimental animals (see Section 6.3.5).

5.11 The curse of the pharaohs

Many of the arguments presented in this chapter were drawn from the report of the Joint Commission [28], and we saw that this report contains clear evidence against nuclear detonations as the cause of ARS in Hiroshima and Nagasaki. We thus might wonder what the commission's members, most of whom were physicians, were really thinking while they assembled their data. The only first-hand account by any of them which I have found is that by the pathologist Averill Liebow [59]. The author offers many interesting glimpses into the conditions of the work, but he does not betray any doubt or puzzlement concerning its scientific findings. However, writing originally in 1965, Liebow notes:

> *It is true that few who took part are left to tell ... Indeed it is as though some curse, like that which the superstitious say fell upon Lord Carnarvon and his men when they violated the tomb of Pharaoh Tut-ankh-amen, has been visited upon those who pried into the ravaged heart of Hiroshima. Only three of the seven American medical officers live. Drs. Oughterson and Tsuzuki, the chief organizers for the two countries, have died; so too, while still young, have Drs. Calvin Koch, Jack D. Rosenbaum, and Milton R. Kramer. May this record do honor to these able and devoted men.*

Liebow's analogy surely is intriguing. We will, however, leave it for others to pursue, lest we be accused of superstition.

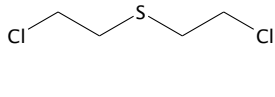
6. Sulfur mustard

Sulfur mustard is a synthetic poison that gained notoriety as the 'king of battle gases' in World War I, in which it caused more casualties than all other poisonous gases combined, even though it was first used only in 1917. Other battle gases like chlorine and phosgene had been used for longer, but their effectiveness had diminished because of protective measures, in particular gas masks. Sulfur mustard bypassed this protection because it attacks the skin, its fumes easily penetrating clothes and sticking to them. By damaging the deeper layers of the epidermis, it causes the formation of blisters, which may become confluent and cause the skin to peel off in large sheets. Agents of this kind are called *vesicants*; the term derives from the Latin word *vesica* (blister). Victims that are not protected by gas masks will also inhale the gas and suffer damage to the the airways; in addition, sulfur mustard may be swallowed and then attack the intestinal tract.

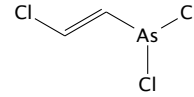
The second most important vesicant is lewisite; it, too, was developed during World War I, but apparently was not deployed. In World War II, both agents were stockpiled by several of the participants, but the only acknowledged use was by Japan in its Chinese campaign. According to Infield [93, p. 187], the U.S. had filled mustard gas into various types of aerial bombs, which were otherwise used for incendiaries; thus, sulfur mustard would have been ready and available for aerial attacks. In the 1980s, sulfur mustard was again used by Iraq in its war on Iran, and its most recent use reportedly occurred in the Syrian civil war [94].

While sulfur mustard and lewisite differ in chemical composition (Figure 6.1), their acute toxic manifestations are very similar [17].[1] For reasons detailed below, we consider sulfur mustard the most likely vesicant to have been used in Hiroshima and Nagasaki, and we will therefore here focus on this agent.

[1] Most sources name sulfur mustard as the poison released in the disaster at Bari, but Maynard [95] in his Master's thesis suggests that it was in fact lewisite. While he presents some intriguing circumstantial evidence, this question is peripheral to the main theme of this book and will not be pursued.



Bis-chlorethyl sulfide
(Sulfur mustard)



Chlorovinylarsine dichloride
(Lewisite)

Figure 6.1 Structures of sulfur mustard and of lewisite

6.1 Physicochemical properties

Sulfur mustard has a boiling point of 217°C and a melting point of 14°C; for deployment at cooler temperatures, the melting point can be lowered by mixing the poison with organic solvents. In its pure form, liquid sulfur mustard is oily and poorly water miscible, which slows down its hydrolysis (decomposition by water). Slow decomposition, a tendency to penetrate porous materials such as wood or bricks, and its high boiling point allow it to persist in the environment for potentially long periods of time. This is illustrated by these words of a British soldier, quoted from Fitzgerald [96]:

> *I suffer badly from phlegm and from coughs and colds a lot. That all started when the British were shelling hard at the last Battle of the Somme. One of the shells disturbed the residue of mustard gas that had been lying there for months. They talk about secondary smoking … I got secondary gas.*

In contrast to sulfur mustard, lewisite has a low boiling point (77°C) and thus is much more volatile; it is therefore likely to dissipate much more readily. We know that the noxious agent used in Hiroshima persisted for weeks [12, 29]; this is the first reason to suspect the use of sulfur mustard rather than lewisite. Another reason is the foul smell, which in Hiroshima was noted by many [11, 12]. Apparently, this smell arises mostly from contaminants in the technical product, which are numerous [97]; the pure product has only a faint smell [98, p. 32]. Lewisite, in contrast, is said to smell only slightly of geraniums [99].

6.2 Mode of action and toxicokinetics

The molecular structures of sulfur mustard and of lewisite are shown in Figure 6.1. Evidently, they are quite different, which suggests that their reactions with molecules within the cells will be different, too, even though the targets and the consequences may be similar.

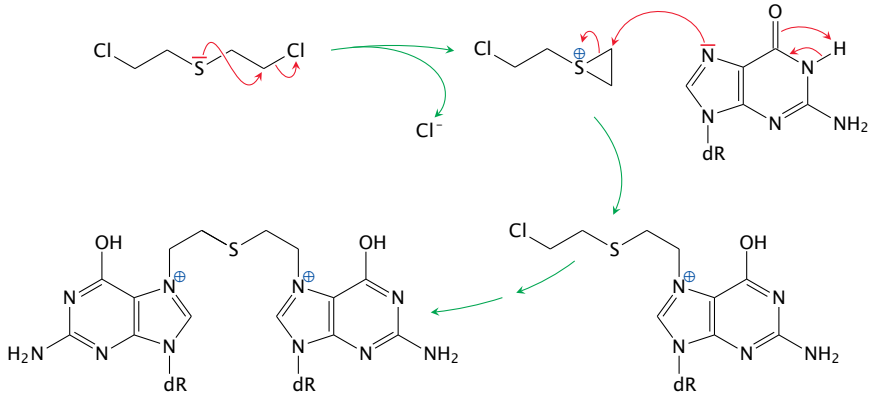


Figure 6.2 Cross-linking of guanine bases in DNA by sulfur mustard. dR represents deoxyribose. The first step consists in the formation of an episulfonium ion; this three-membered ring is highly reactive and readily attacked by the N7 of guanine or by other nucleophiles such as glutathione. Capture of the second guanine involves the same steps as shown explicitly for the first one.

6.2.1 Reaction with DNA. The reaction of sulfur mustard with DNA begins with the formation of an episulfonium ion (Figure 6.2). This three-membered ring is highly unstable and may react with any nucleophiles within the cell; but, for the same reasons as with ionizing radiation (Section A.11), the most consequential target molecule is DNA. Any of the four bases found in DNA[2] may react, but the most reactive one is guanine, and in particular the specific nitrogen (N7) in the imidazole ring shown in the Figure. Importantly, sulfur mustard is a *bivalent* molecule; both of the two chloroethyl ($-CH_2-CH_2-Cl$) groups attached to the central sulfur atom can react in the same manner. This may cause the formation of a cross-link between two bases on opposite strands of the DNA molecule; and downstream of such cross-links, both strands may break,[3] resulting in the same kind of lesion also observed with ionizing radiation. An important role of such cross-links in the biological effect of sulfur mustard is supported by the early finding that similar compounds in which one of the two reactive groups is missing have much lower toxicity [98, p. 35].

The similarity of the mutagenic DNA lesions caused by ionizing radiation and by sulfur mustard explains that both noxious agents produce similar biological effects both in the short term, such as bone marrow damage, and in the long term, such as leukemia and cancer. The reactivity of lewisite toward DNA has

[2] These bases are the purine derivatives adenine and guanine, as well as the pyrimidine derivatives cytosine and thymine. Within RNA, uracil replaces thymine.

[3] This has been demonstrated with nitrogen mustard [100], which reacts with DNA in the same manner as does sulfur mustard.

received surprisingly little attention; unlike sulfur mustard, however, lewisite has no clearly documented mutagenic or carcinogenic potential [17, 101]. The significantly increased incidence of leukemia and of some solid tumors among survivors of the Hiroshima and Nagasaki bombings [102, 103] thus further supports the thesis that sulfur mustard rather than lewisite was used in the destruction of both cities.

6.2.2 Depletion of glutathione. While reaction with DNA mediates most of the damage at low concentrations of sulfur mustard, reactions with other nucleophiles provide an alternate mechanism of toxicity at higher levels. A particularly important molecule is glutathione, which has a key role in scavenging many kinds of toxic compounds inside the cell. If glutathione is depleted by its reaction with sulfur mustard, this will impair the cell's ability to neutralize *reactive oxygen species* (ROS), which arise as main or side products of many metabolic processes; the unscavenged ROS may then cause cytotoxic effects [104].

One biochemical pathway that involves ROS is the formation of skin pigment (melanin); and the melanocytes (pigmented cells) of the skin, which carry out this pathway, are more susceptible to sulfur mustard toxicity than are the keratinocytes (non-pigmented cells; [105]). Accordingly, levels of exposure that kill the melanocytes yet permit the keratinocytes to regenerate may cause skin depigmentation. On the other hand, lower levels of sulfur mustard that permit both keratinocytes and melanocytes to regenerate may result in hyperpigmented skin areas.

Glutathione reacts with sulfur mustard via its sulfhydryl (–SH) group, which makes an excellent nucleophile for attacking the episulfonium intermediate shown in Figure 6.2. Although the chemistry is different, sulfhydryl groups also react strongly with lewisite; this suggests that indeed the similarity of the early manifestations on skin and mucous membranes is mediated by this mechanism. Experimental data on the reactions of lewisite *in vivo* are, however, very sparse [17, 101].

6.2.3 Systemic uptake and distribution. Sulfur mustard is taken up through skin contact, inhalation, and ingestion. Soldiers exposed to sulfur mustard in World War I, as well as the workers in the factories producing the poison, were often protected by gas masks; aware of the danger, they would mostly have avoided ingestion of contaminated food or water. In contrast, the unprotected and unaware victims in Hiroshima and Nagasaki most likely took up significant amounts by all three routes.

When applied experimentally to the skin of experimental animals, 80% of the compound will typically evaporate, but the other 20% will be taken up. Approximately 80% of that latter fraction, or 16% of the total, will indeed reach the blood circulation and then the inner organs, while the remainder (4% of the total) will react and remain in the skin [106]. The fraction taken up into the system distributes between different organs. While the relative abundance found in different organs differ somewhat between studies that use different methods of detection—chemical [107], radioactive tracer [108, 109], or DNA damage [110]—it is apparent that organs with strong blood flow receive and retain the highest amounts. These organs include the brain, the lungs, the spleen, and the kidneys.

As noted earlier, sulfur mustard is poorly water-miscible; such substances are *hydrophobic* or *lipophilic*, and they tend to accumulate in lipids (fat-like substances). The brain is not only strongly perfused, but also particularly rich in lipids in the form of *myelin*, which enwraps many nerve fibers and serves as an electrical insulator. It is therefore understandable that the highest abundance of DNA adducts is found in the brain [110], slightly ahead of the lungs. However, since cell proliferation in the brain is generally very slow, this organ is not very sensitive to the consequences of DNA damage by sulfur mustard; this parallels its low susceptibility to radiation.

With the passage of time, sulfur mustard will redistribute from the brain and other organs into the tissue with the highest fat content—fat tissue. This was demonstrated by Drasch et al. [107], who examined the body of an Iranian soldier who had succumbed to sulfur mustard poisoning one week after exposure. It is notable that the sulfur mustard observed after this time was still in its native, unreacted form. Slow redistribution, via the bloodstream, from fat tissue to other organs would likely give rise to protracted DNA and cell damage over time; this may contribute to the oft-noted slow recovery of sulfur mustard victims, and also to the delayed onset of 'radiation sickness' in patients from Hiroshima and Nagasaki xref.

Among the cited studies, only [108] gives levels of sulfur mustard and of DNA adducts for the bone marrow, which are surprisingly low. Nevertheless, the susceptibility of the bone marrow to sulfur mustard is a long-established fact [111], as is that of the gonads. Overall, we note that high levels are reported consistently in some organs—brain, lungs, and kidneys—that are among the least susceptible to ionizing radiation.

6.2.4 Metabolism. The reactive nature of sulfur mustard makes it susceptible to several pathways of metabolic conversion and inactivation. We already mentioned the reaction with glutathione; this reaction is facilitated by the enzyme

glutathione-*S*-transferase, which is particularly abundant in the epithelial cells of the liver and the small intestine. Glutathione conjugation is an effective detoxification pathway for drugs and xenobiotics; as long as glutathione is not depleted by large amounts of substrate—such as, for example, sulfur mustard in the skin—this reaction is beneficial.

Sulfur mustard is also susceptible to hydrolysis, which occurs in two steps and results in its inactivation:[4]

$$\mathrm{Cl{-}(CH_2)_2{-}S{-}(CH_2)_2{-}Cl + H_2O \longrightarrow Cl{-}(CH_2)_2{-}S{-}(CH_2)_2{-}OH + HCl} \quad (6.1)$$

$$\mathrm{Cl{-}(CH_2)_2{-}S{-}(CH_2)_2{-}OH + H_2O \longrightarrow HO{-}(CH_2)_2{-}S{-}(CH_2)_2{-}OH + HCl} \quad (6.2)$$

Another important reaction is oxidation, which occurs extensively *in vivo* [112]. The enzymes responsible have apparently not been characterized; until such evidence becomes available, both cytochrome P450 and peroxidase enzymes are plausible candidates. The first oxidation intermediate is the sulfoxide, which has low toxic activity (Figure 6.3); however, a second oxidation will give the sulfone, which can eliminate HCl and thereby turn into divinyl sulfone, which is highly reactive and mutagenic [113]. In this context, it is noteworthy that a high level of peroxidase occurs in the thyroid gland. Thyroid peroxidase is known to mediate sulfoxidation of structurally similar thioether compounds [114], and conversion of sulfur mustard to divinyl sulfone in the thyroid gland might expose this organ to increased carcinogenic activity. Thyroid cancer has been observed in Iranian sulfur mustard victims [115], and its incidence is also significantly increased in Hiroshima and Nagasaki survivors [116].

6.3 Clinical and pathological manifestations

From its biochemical mode of action, it is clear that sulfur mustard is not selective for any organ or cell type. Therefore, the extent of damage to any particular organ is largely governed by the extent of its exposure. Directly exposed are usually the skin, the eyes, and the airways and lungs. The fraction of the poison that is taken up will preferentially affect organs that are strongly perfused, such as the lungs, the brain, the spleen, the kidneys, as well as the adrenal and thyroid glands. In organs exposed to high doses, glutathione depletion is more likely to cause damage in the short term; in those subjected to lower doses, the tendency to respond to DNA damage with apoptosis (programmed cell death) is a crucial determinant. The latter category includes in particular the gonads, the bone marrow, and the lymphatic tissues.

[4] Hydrolysis will also occur in the environment; however, since sulfur mustard is poorly water-miscible, this process will be slow.

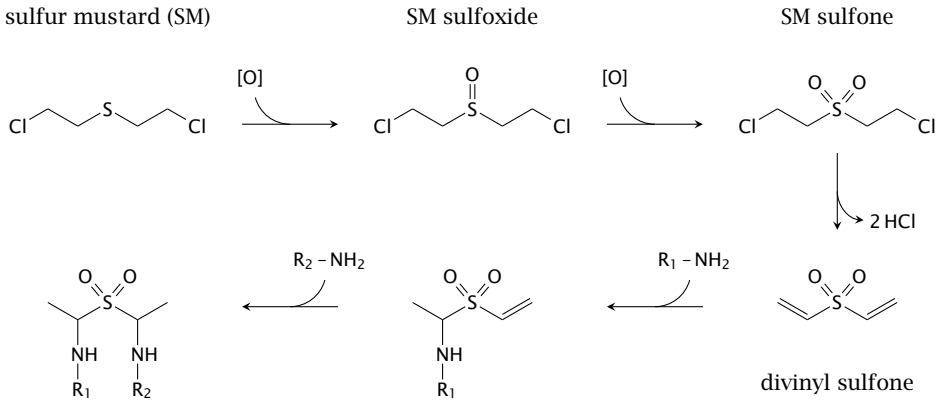


Figure 6.3 Oxidative metabolism of sulfur mustard. The sulfur atom may become oxidized, most likely by peroxidase or monooxygenase enzymes, to give first the sulfoxide and then the sulfone, which can eliminate HCl to yield divinyl sulfone, which like native sulfur mustard has two reactive groups and is mutagenic.

Most organs will become exposed to sulfur mustard through the blood circulation; and since the blood levels are evidently high enough to cause severe damage in multiple organs, we can also expect toxicity to the blood circulation itself.

6.3.1 Blood circulation. In experimental animals exposed to sulfur mustard, the larger blood vessels (arteries and veins) were observed to lose tone and become dilated; the affected organs will appear increasingly filled with blood (congested). The smallest blood vessels (the capillaries) became leaky; plasma fluid and proteins were lost from the bloodstream, as sometimes were blood cells, and caused the surrounding tissues to swell [24].[5] Such findings explain the clinical picture of hypovolemic shock and general edema in severely exposed victims [117] or experimental animals [13]. Leakiness of the microcirculation is also apparent from the loss of plasma proteins in the urine; and acidity of the urine indicates metabolic acidosis, which is typical of severe circulatory shock [98, p. 228].[6] While the poisoned victims will initially look pale, as perfusion of the skin is largely shut off in favor of the vital organs, in later stages they

[5]The hydrostatic pressure in the capillaries always exceeds that within the surrounding tissue. Normally, this pressure gradient is balanced by the osmotic effect of the large quantity of protein contained in the blood plasma. Once the capillary walls become leaky toward the plasma proteins, however, this balancing mechanism breaks down, and plasma freely seeps into the tissues. Any fluid added through drinking or infusion will do likewise and amplify the edema.

[6]Shock, in the pathophysiological sense, is the failure of the circulation due to lack of blood volume, to loss of vascular tone, vascular leakage, or to failure of the heart.

will appear swollen and cyanotic. The loss of plasma fluid should also trigger intense thirst; this is documented in cases of severe mustard gas poisoning [98, p. 228], and it is also typical in other diseases that cause generalized leakiness of the microcirculation, or *capillary leak syndrome* [118]. Even with intensive care readily available, this condition is often fatal [119], and it will of course be even more so under field conditions.

The proteins contained in the extravasated plasma fluid include coagulation factors and fibrin, which will become activated and may solidify. Particularly in the lungs, this can result in the formation of fibrin 'casts' that obstruct the lumen of the bronchi and bronchioli; this is observed both in autopsies of human victims [24, 98] and experimentally [120].

6.3.2 Airways and lungs. In mustard gas victims not protected by gas masks, the airways and lungs are prominently affected. The inhaled sulfur mustard will condensate atop the mucous membranes and attack the epithelial cells within them; the necrotic cell layers may remain in place, held together by coagulated fibrin, as so-called *pseudomembranes* [121], or they may desquamate in a manner similar to the epidermis of the skin. Either way, the victims will experience hoarseness and pain in the throat and chest, and they will have difficulty breathing and swallowing.

The bronchi may become obstructed by edema and by fibrin extravasation and cast formation (see above), or by coagulated blood spilling from damaged blood vessels[120]. Coagulation can also be activated within the lung's blood vessels themselves, causing thrombus formation and flow obstruction [122]. Since partially obstructed bronchi tend to let more air in than out, air will become trapped in the peripheral lung tissue, a condition known as *emphysema* [24]. Distended zones of lung tissue will then compress adjacent ones and disrupt their ventilation. Elevated pressure and structural injury may permit the trapped air to leave its regular confines and enter the interstitial space of the connective tissue; this is referred to as *interstitial emphysema*.

If the patient survives this initial stage, the inured lung tissue will be susceptible to infections, and thus foci of bronchopneumonia will develop. Overall, lungs damaged by sulfur mustard will exhibit general circulatory congestion and a varied pattern of bronchial obstruction, hemorrhage, and inflammation.

6.3.3 Eyes. Affliction of the eyes is usually early and painful (Figure 6.4), but also transient. The lesions to the exposed parts of the eyeball, the cornea and the conjunctiva, are similar in principle to those found on the epidermis and mucous membranes, with necrosis and desquamation; however, they are mitigated by the prompt and steady rinsing action of the tear fluid.

Figure 6.4 Ocular symptoms of mustard gas exposure, A: Eyelid edema and blepharospasm in a sulfur mustard victim one day after exposure, which occurred in 2016 in Syria. Skin desquamation with secretion and blisters are also seen. Reproduced from Kilic et al. [94] with permission by the corresponding author (Mesut Ortatatli). B: British soldiers in World War I, transiently blinded by exposure to sulfur mustard. Photograph by Second Lieutenant T. L. Aitken; Imperial War Museum, London.

The corneal epithelium, when damaged, will initially appear turbid and then erode; this causes impaired vision, pain and reflexive blepharospasm. In combination, these symptoms will create a subjective perception of blindness; Alexander [18] reports that some of his patients believed themselves permanently blinded until their eyes were forced open to prove that this was not so. The deeper layers of the cornea, and the remainder of the eyeball, may escape undamaged. The eroded epithelium will regenerate from the periphery toward the center. In most cases, the loss of vision is reversible within days or a few weeks.

While the above covers the consequences of external exposure, it is also necessary to consider the possible effects on the eyes of sulfur mustard transported in the bloodstream. While the literature offers no pertinent experimental evidence on sulfur mustard itself, some studies have been reported on various functionally similar compounds, including nitrogen mustard and busulfan, which are or were used in the treatment of cancers and leukemias. Patients thus treated may develop symptoms in parts of the eyeball not usually affected by superficial exposure: cataract, which afflicts the lens; *uveitis*, that is, inflammation of the iris and adjacent soft tissue structures; and edema of the retina has been described as well [123]. Cataract has been induced with nitrogen mustard and busulfan in experimental animals also [124, 125], and a similar effect seems likely after systemic uptake of sulfur mustard. In addition, we can expect bleeding in the retina and other places in patients with generalized purpura due to bone marrow suppression xref.

6.3.4 Skin. While skin blisters are a prominent feature of mustard gas lesions, the spectrum ranges from erythema only over desquamation and blisters to deeper necroses of all layers of the skin and the underlying soft tissues. The severity will vary not only with the amount of sulfur mustard applied, but also with the texture of the skin and its humidity; the palms of the hand have thicker skin and are less susceptible, whereas areas covered by tender and humid skin such as the armpits are more so. Severe lesions may be surrounded by a halo of less severely afflicted areas. When such lesions heal, the more lightly affected peripheral areas tend to become hyperpigmented (Figure 6.5), whereas the more severely affected ones will show depigmentation. The reason for this was discussed in Section 6.2.2 above.

The skin may be exposed by being splashed directly with liquid sulfur mustard, but also by indirect contact with contaminated weapons or other objects, as well as by the fumes, which easily penetrate clothes, even in multiple layers. While mustard splashed on exposed skin areas may be rapidly wiped and washed off before doing much damage, contaminated clothes may function as a

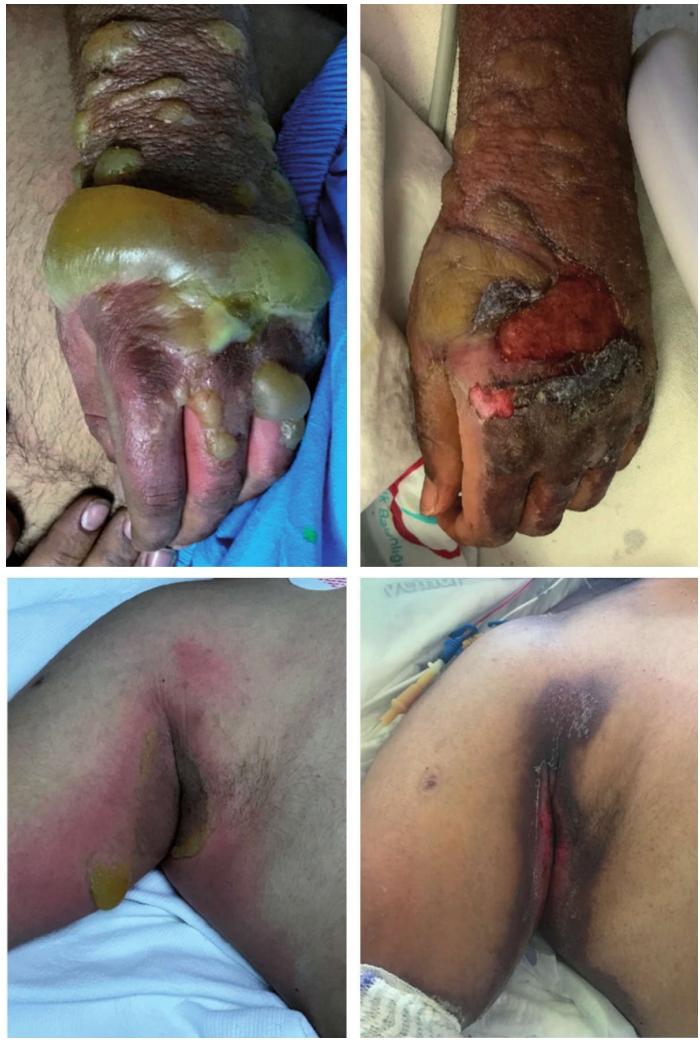


Figure 6.5 Skin lesions in mustard gas victims. Top: large blister in an early lesion, and beginning wound healing after partial removal of dead tissue at a later stage. Bottom: axillary lesion, surrounded initially by erythema and later on by hyperpigmentation. After 11 days (left), necrotic skin is still adherent; it is sloughed off several days later (right). Reproduced from Kilic et al. [94] with permission by the corresponding author (Mesut Ortatatli).

reservoir of the poison and cause more severe damage to the skin underneath. Examples of skin lesions observed underneath clothing are shown in Figure 6.6. Similarly, Alexander [18] reports that, among the mustard victims at Bari, those that stripped off their contaminated clothes of their own initiative fared much

better than those who kept them on for the night following the disaster. Such apparent negligence can be understood only if we consider that the onset of mustard skin lesions is typically delayed by several hours; once the pain becomes perceptible, the poison has already been taken up, and the damage is done. On the time course of the clinical manifestations, the American military physician Harry Gilchrist notes [126, p. 44]:

> *At first the troops didn't notice the gas and were not uncomfortable, but in the course of an hour or so, there was marked inflammation of their eyes. They vomited, and there was erythema of the skin. ... Later there was severe blistering of the skin, especially where the uniform had been contaminated, and by the time the gassed cases reached the casualty clearing station, the men were virtually blind and had to be led about, each man holding on to the man in front with an orderly in the lead.*

A careful experimental study on the time course of mustard skin lesions Pullinger [127] also documents a slow, gradual progression. They early stage consists in a massive edema through extravasation, indicating capillary damage. Blood flow remains intact for several days, even though necrosis of the tissue is underway; vascular occlusion and sequestration of necrotic tissue finally occur after some 10 days. Such a time course resembles clinical observations.

6.3.5 Digestive tract. The earliest and most common gastrointestinal symptom is vomiting. Unless it is bloody, however, vomiting need not be due to direct action of the poison on the digestive organs, but may instead result from its stimulation of the *area postrema* in the brainstem, which triggers vomiting in response to various chemical agents. More specific indications of damage to the intestinal organs themselves is diarrhea, which in severe cases may also be bloody.

Warthin and Weller [98] relate that physicians who had been treating cases of mustard poisoning in World War I disagree as to whether diarrhea constitutes an early and typical symptom of mustard gas poisoning. Two cases described in detail by Heitzmann [24] developed diarrhea only about 10 days after the exposure; on the other hand, Warthin and Weller [98, p. 75] describe an acute case with acute diarrhea, together with vomiting, and they also find report rapid onset in experimental animals injected with the poison (pg. 91). Dacre and Goldman [13], too, cite a number of experimental studies on animals and human case reports that list early diarrhea as a typical symptom of mustard gas poisoning.

Whether or not diarrhea occurs in a given case of mustard gas poisoning may simply depend on the dosage. The digestive tract may receive mustard gas

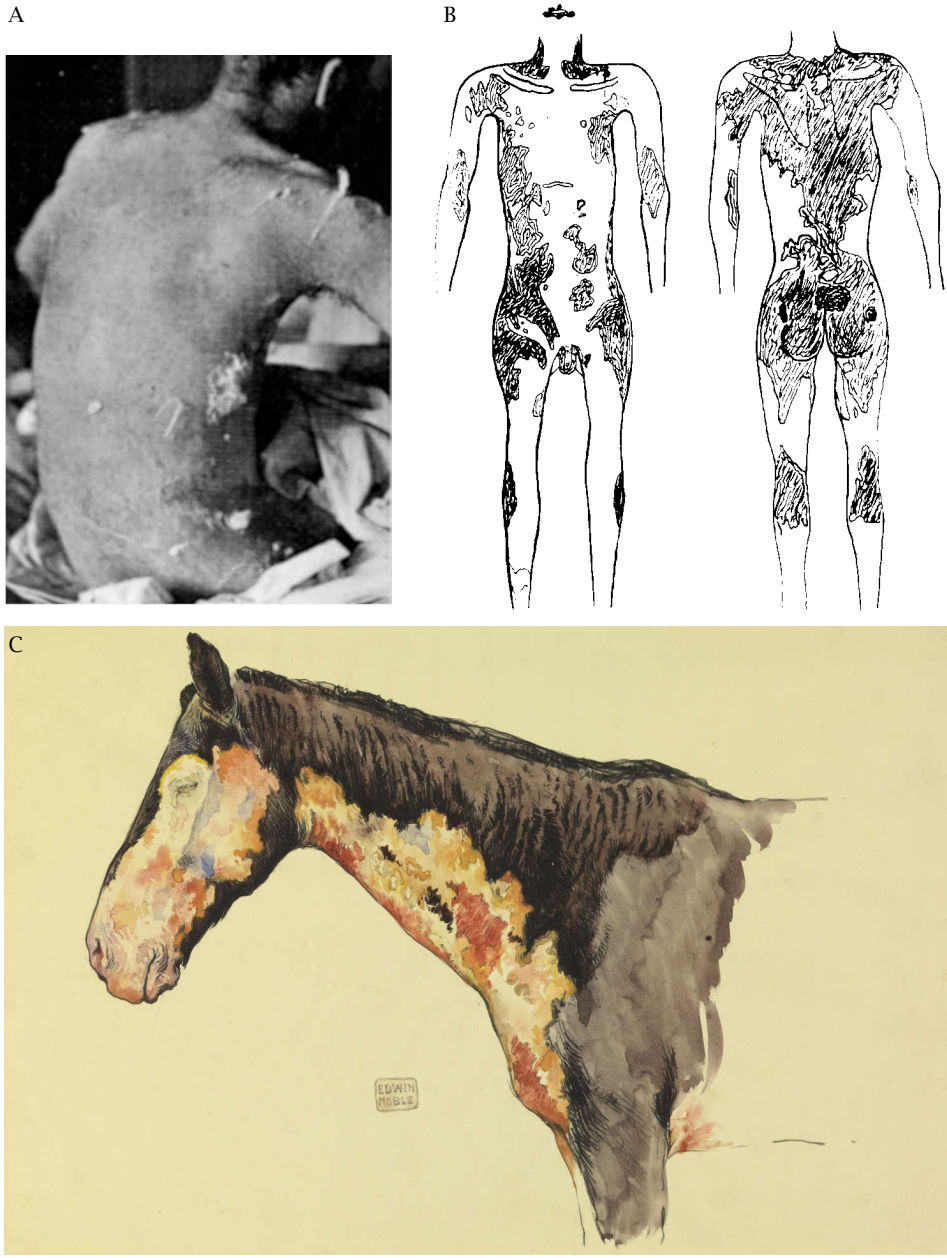


Figure 6.6 Clothes or hair do not protect from mustard gas. A: Fraying and desquamating skin in a mustard factory worker. B: Distribution of lesions in another affected worker. A and B adapted from [98]. C: Skin lesions in a warhorse exposed to mustard gas in World War I. Sketch by Edwin Noble (Imperial War Museum, London).

both by ingestion and through the bloodstream. In the first case, one would expect higher local levels and earlier onset of symptoms, whereas in the latter case levels in the GI tract may be lower and the onset of manifest symptoms delayed, as is the case with the bone marrow.

Autopsy reports paint a somewhat variable picture, with edema, focal or regional necroses, pseudomembranes, hemorrhages within the mucous membranes or spilling out into the lumen, and secondary infections. Overall, the pathological features are rather similar to those observed in the respiratory tract.

6.3.6 Bone marrow, spleen, and gonads. These organs host cell types that are highly susceptible to radiation, and they likewise are highly susceptible to the genotoxic effect of sulfur mustard. In many cases, it is indeed the bone marrow toxicity that causes the patient's demise, through either uncontrollable bleeding due to lack of thrombocytes, or uncontrollable infections due to the lack of leukocytes. Accordingly, in the autopsies of such patients, one finds a barren bone marrow, absent sperm cell production, and depletion of lymphocytes in the spleen. As far as I can tell, none of these observations allows one to discriminate organ damage by sulfur mustard from that through radiation.

6.3.7 Kidneys, liver, and brain. In most cases, these organs show signs of damage to the vascular system rather than to the organ-specific epithelial or nerve cells. The blood vessels are congested, occasionally bleeding into the tissues has occurred; in the liver, there may be signs of slight fatty degeneration, and in the kidneys protein may have seeped out of the blood vessels, into the urine-conducting and -processing conduits (the *tubuli*; [24]). These changes, while not overly dramatic, are not expected in patients exposed to doses of radiation that do not kill on very short notice (1-2 days).

6.3.8 OK, wer hat noch keinen Fahrschein?. We haven't talked about the hair falling out, I guess. Anything else?

7. Burns

Disfiguring scars of the skin have a prominent place in the story of Hiroshima and Nagasaki. These lesions are mostly ascribed to the 'flash burns' caused by light from the 'ball of fire,' which is said to have formed during the first second of the nuclear detonation [128]. One might wonder why, among the various physical effects accompanying a nuclear detonation, only the flash of light is considered in this context. Can we rule out ionizing radiation as a possible cause of skin burns?

When animals are experimentally irradiated with γ-, X-, or neutron rays at doses that are lethal due to their effect on the bone marrow or other sensitive organs, the skin nevertheless shows little evidence of injury [22, p. 44 ff.]. Thus, if someone survives a nuclear detonation by 20 days or beyond, as is the case with the group of victims surveyed by the Joint Commission [28], we can infer that any major skin burns could not have been caused γ- or neutron rays from the bomb. Preferential damage do the skin could indeed be brought about by β-rays (see Section A.7.1 and [22]); and some β-radiation would indeed have been given off by radionuclides in the fallout. However, the observed low levels of radioactivity in the fallout could not have caused acute injury.[1] Thus, the only mechanism that remains for the causation of skin burns by nuclear bombs is indeed thermal radiation.

It is worth noting that a nuclear detonation releasing a flash of light as intense as claimed to have occurred in the bombings should indeed have caused flash burns. This is confirmed by experimental studies, some of which are discussed in Section 7.6. However, as we will see in this chapter, many features of the observed burns show that they cannot in fact have been such flash burns; the evidence points instead to napalm and to mustard gas as the true causes of the burns.

[1] Yamada and Jones [80] report 'obvious' effects of high β-doses in a relatively small group of Hiroshima victims who had been exposed to black rain. However, these authors don't report skin burns, but instead base their claim on epilation and mucosal symptoms; and they disregard that these victims also exhibit purpura, which is a clear sign of bone marrow damage and could only have been caused by more penetrating forms of radiation.

7.1 Classification of skin burns

Before we get to the evidence, a few words about terminology are in order. Skin burns can be classified according to the cause and, independently, according to severity.

7.1.1 Causes of burns. These include contact (hot objects or liquids, napalm), chemicals (sulfuric acid, mustard gas), and thermal radiation. Although all of the major causes that we will consider here—flash burns, napalm, and mustard gas—fit into this classification, they all differ from more commonplace causes encountered in civilian life.

Mustard gas burns develop more slowly than those with most other chemicals, such as strong acids (sulfuric or hydrochloric acid) or bases (lye). The delayed onset of its effect makes mustard gas particularly treacherous. This is illustrated by the casualties of the Bari incident (Section 1.4.4): the victims did not perceive any pain shortly after exposure, and many neglected to change their contaminated clothes before the night, only to awake to severe skin burns on the next day [18].

Napalm burns may be classified as contact burns. However, in this case the combustible material is designed to stick together in sizable chunks that adhere to target surfaces [129], which means that the amount of heat transferred to those surfaces will be unusually high. Thus, compared to conventional contact burns, napalm burns tend to be particularly severe [130, 131]. (xref to napalm section or chapter.)

Nuclear flash burns are a special case of burns caused by thermal radiation. Here, the energy is delivered in a particularly brief and intense pulse, which means that the heat absorbed by the skin has no time to dissipate toward the tissues beneath, but instead causes very high temperatures within a thin superficial layer. Investigators have found ways to emulate such high intensity flashes; some results of such studies are detailed in Section 7.6.

7.1.2 Severity of burns. This is expressed in degrees:

- first degree burns show irritation and erythema (reddening), but no damage to the anatomical skin structure;
- in a second degree burn, a superficial layer of the skin detaches to form a blister. Usually, the skin underneath can regrow from deep-set patches within hair follicles or glands and heal quickly, with minor scarring or without it;
- a third degree burn destroys the entire depth of the skin. The wound is closed by new skin growing inwards from the periphery, and a scar will form;
- a fourth degree burn includes significant injury of tissues beneath the skin.

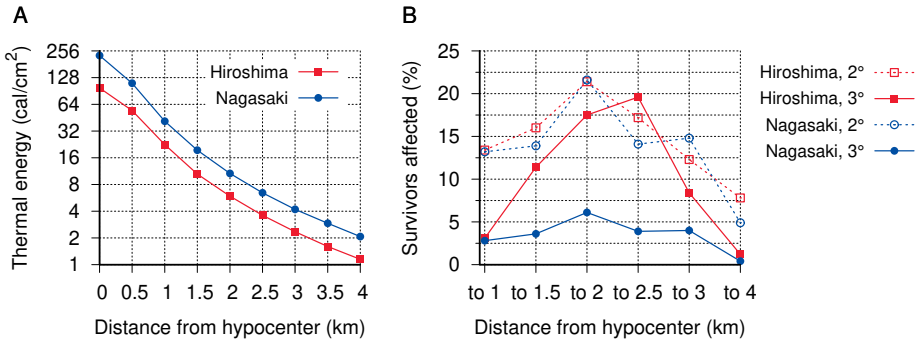


Figure 7.1 Radiant heat and incidence of burns as functions of distance from the hypocenters at Hiroshima and Nagasaki. A: radiant heat (calories per cm^2) vs. distance from hypocenter. Data from Ishikawa et al. [7]. B: Incidence of second and third degree burns in victims remaining alive after 20 days, by distance from the hypocenter. Patients with both second and third degree burns are counted only among the latter. Data from [28, Tables 9H and 9N].

All manifest burns should be painful to some degree, even though the intensity and the quality of the pain may vary. Volunteers who received experimental first or second degree flash burns uniformly reported instantaneous pain (see Section 7.6). Third and fourth degree burns will destroy the nerve endings of the skin together with the skin itself, which may alter pain quality and intensity; however, as long as the victims remain conscious, they should still perceive some sort of pain, originating from pain receptors in the most superficial layers of tissue that remain viable. With chemical burns, however, pain will often not be perceived in the instant of contact with the chemical, but only after the chemical has penetrated the skin and a damaging chemical reaction has had time to occur. As noted above, with mustard gas in particular the manifestation of visible lesions and the perception of pain tend to be delayed.

7.2 Statistical observations on burns in Hiroshima and Nagasaki

7.2.1 Flash burns vs. flame burns. When the survivors studied in each city by the Joint Commission (see Section 5.4) were grouped by distance from the hypocenter [28, Tables 8H and 8N], up to 47.3% displayed burns of any kind. Up to 1.4% of all victims were diagnosed with only flame burns, and up to 32.6% with only flash burns. Up to 7.3% were listed with both flame and flash burns, while with up to 9% the type of burn was stated as unknown. Thus, the majority of burns were considered flash burns, but the presence of some putative flame burns must be kept in mind.

7.2.2 Observed incidence of burns by distance from the hypocenter. Figure 7.1A shows the intensity of the thermal radiation as a function of distance from the hypocenters.[2] Within 1 km of the hypocenter, these intensities would have exceeded anything that has been tried in experiments on human volunteers or animals (see Section 7.6); however, we can extrapolate that such doses should cause burns of at least third degree. Overall, considering the postulated intensities and the experimental findings, we should expect the following features in the distribution of flash burns about the hypocenter:

- the number and severity of flash burns should have been greatest near the hypocenter. With increasing distance from it, both incidence and severity should have decreased;
- within 1 km of the hypocenter, most flash burns should have been of third or fourth degree. Lower degrees should only have occurred with attenuation by at least two layers of clothing or some equivalent partial protection;
- burns should have been more severe in Nagasaki than in Hiroshima, or at least not less so.

Figure 7.1B shows that none of these expectations corresponds to observation. The incidence of third degree burns grows from the hypocenter towards a maximum at 2 or 2.5 km, respectively. In Hiroshima at least, this increase is so pronounced that it cannot plausibly be explained by the statistical noise from flame burns.[3] To judge burn severity, we can look at the ratio of third degree burns to second degree burns. In Hiroshima, this ratio also increases substantially between 1 and 2.5 km. In Nagasaki, neither trend is as pronounced, but both the incidence of third degree burns and the burn severity are strikingly lower than in Hiroshima, even though the bomb yield, and therefore the thermal radiation, are said to have been greater in Nagasaki.

7.2.3 Flash burns in skin areas covered by clothes. Clothes should afford partial protection from flash burns (see Section 7.6). Since dark clothes will absorb heat more readily than white or light ones, we might expect flash burns in covered areas to be more common with dark clothes. The numbers stated in [28, Table 13] support such a relationship: those wearing colored clothes

[2] The table (…) in the source contains, for each city, two slightly different estimates for different assumed visibilities, of which Figure 7.1 A shows the averages.

[3] If we ascribe all third degree burns to patients with flash burns only within 1 km, but the minimum possible number between 2 and 2.5 km, then the incidence of third degree burns in patients with flash burn only drops is 22.3% within 1 km and remains at 22.1% between 2 and 2.5 km. Thus, even this extreme scenario fails to show the expected decrease in burn severity.

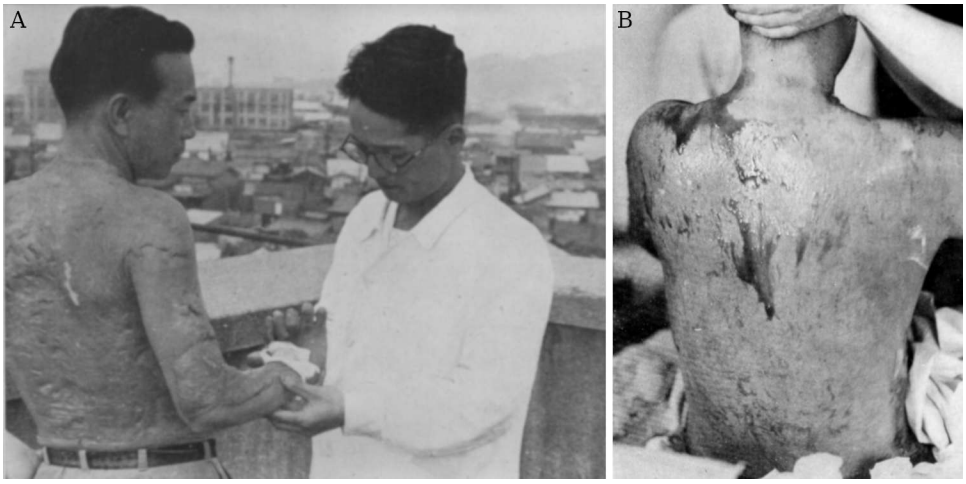


Figure 7.2 Burns of the skin limited to areas that had been covered with clothing. A: keloids subsequent to 'flash burn' in a bombing victim receiving treatment at Hiroshima's Red Cross Hospital, several years after the bombing [132]. The physician pictured is Dr. Terufumi Sasaki, who is portrayed in John Hersey's book *Hiroshima* [6]. B: chemical burn in an American mustard gas factory worker [98].

more often had burns in covered areas in addition to uncovered ones.[4] With neither white nor colored clothes, though, we would expect any burns to occur in the covered areas *only*, without any burns in the exposed skin. However, the scars left by just such a burn are seen in Figure 7.2A. The scars cover almost the entire upper body and the arms of the victim, but none are visible above the collar line. A strikingly similar distribution is observed in panel B, which shows a victim of mustard gas exposure; we note only some dark pigmentation, but no deep lesions on the back of the neck.[5] For further examples of the same effect in alleged nuclear flash burns, see [59, 133, 134]. Moreover, Oughterson et al. [28, Table 13] state that 5.4% of all burn victims in Hiroshima, and 9% of those in Nagasaki, had burns in the clothed area only.

[4] This is a rare example of an observation that is indeed most readily explained by the orthodox story of nuclear detonations, which I urge its believers to duly celebrate. However, these burns are not grouped by distance from the hypocenter; colors may have differed between inner city and surrounding districts. The number of layers of clothes in either group is also unknown.

[5] The mustard-exposed patient in the picture was initially treated with oil-based unguents ('the grease method'), which resulted in gangrenous infection; he improved after his treatment was switched to aqueous disinfectants. Father Arrupe, a Jesuit priest and physician who cared for a number of burned patients in Hiroshima, thought that the oil treatment administered by Japanese physicians promoted infections and the formation of keloids [132].

Whatever the color or thickness of the clothes, they would have to be burned away by the radiant heat first in order to reach the skin underneath. Nevertheless, some burns apparently occurred underneath the intact clothing. Eyewitness Mr. Hashimoto relates giving first aid to a girl with burns on her backside, as quoted by Hachiya [47]:

> *I . . . began painting [with mercurochrome] the wounds of a girl dressed in* monpe *[pants] . . . Her wounds were mostly on her buttocks and these I found hard to bandage, for when she stood up the bandage slipped off. . . . Finally, I gave up and in desperation pulled down her monpe, and after repainting her wounds, pulled up her monpe and put the bandages on right over them.*

From this account, it is quite clear that this girl still *had* her pants, yet had suffered burns underneath them, in a location that is commonly affected by sulfur mustard, as moist skin areas generally are (see Figure 6.6 and [98]).

Finally, while I have not seen any experimental studies on the subject, I surmise that the layer of sturdy hair that covers the skin of a horse should provide substantial protection from flash burns. Nevertheless, there are multiple reports of horses having suffered burns as well, for example this one by eyewitness Akihiro Takahashi [76, p. 193]:

> *. . . a horse, only raw flesh, lying dead with its head in a cistern.*

While we cannot be sure about the cause of such burns in each single instance, a plausible one is mustard gas, which should penetrate hair and fur just as readily as it penetrates clothing. Mustard gas lesions in horses were indeed noted in World War I (see Figure 6.6). Overall, therefore, the manifestations of burns in covered skin observed in Hiroshima and Nagasaki do not fit the pattern expected of true flash burns.

7.2.4 Irregular shapes of flash burns. Much like a sunburn, a flash burn should affect the exposed areas of skin quite evenly. Figure 7.3A shows the expected distribution; however, part of the skin shows fresh erythema, even though this picture was taken only on October 11th, that is, more than two months after the bombing. While experimental flash burns of light or moderate severity indeed initially manifest as erythema, they progress within days either to heal without defect, or to first shed the damaged skin and then heal, possibly with some degree of scarring (see Section 7.6). Thus, the erythema visible on October 11th could not have been caused by the bombing on August 6th. We can speculate, but cannot prove, that this fresh sun burn was staged and photographed as

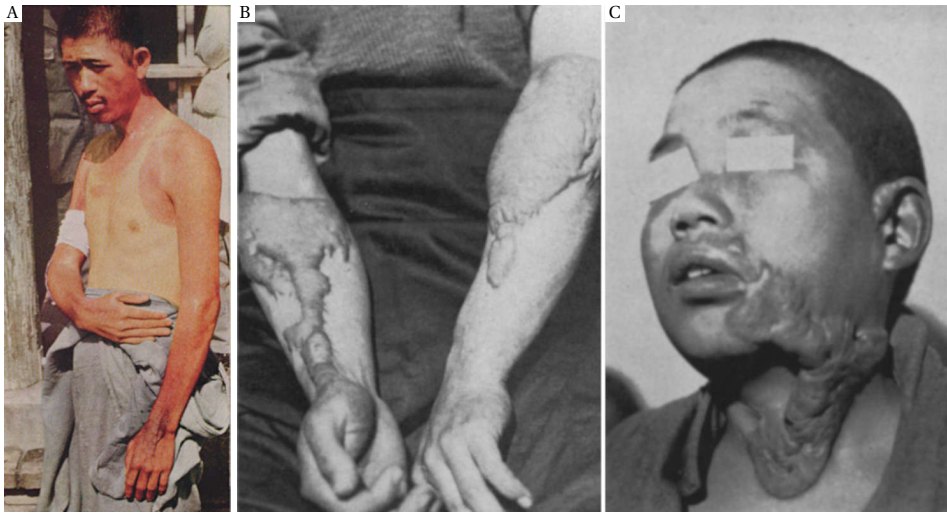


Figure 7.3 Skin lesions in Hiroshima bombing victims ascribed to 'flash burn'. A: general erythema and local hyperpigmentation of exposed skin in a man exposed at 2.4 km from the hypocenter; photographed on October 11th 1945. Taken from Oughterson and Warren [78, p. 147]. B and C: keloids (hypertrophic scar tissue) in two patients exposed at 1.3 and 1.7 km, respectively, from the hypocenter. Taken from Block and Tsuzuki [133].

a welcome present for the Joint Commission that arrived in Hiroshima on the following day.[6]

Panels B and C of Figure 7.3 show keloid or hypertrophic scar tissue formed in lesions ascribed to flash burns.[7] The lesions have highly irregular shapes that cannot plausibly be explained with any sort of partial cover by clothing or shielding. Nevertheless, such irregular shapes are typical of 'flash burn' illustrations in both general and medical references; the more regular pattern shown in panel A is the exception. The irregular shape was noted by early observers. Shigetoshi Wakaki, a Japanese military officer who was involved in weapons research and development, and who entered Hiroshima shortly after the bombing, notes [135, p. 88]:

[6]The reference from which this photograph is taken [78] claims it to show 'pigmentation', but pigmentation is pronounced only on the wrists, whereas on most of the arms it is suggestive of a sun tan. Much of the visibly colored skin is red, not brown; and the authors, both ivy league professors of medicine, were surely aware that humans don't produce red skin pigment.

[7]There is some variety of opinion on whether or not keloids are the same as hypertrophic scars. The reference from which these pictures were taken [133] lumps them together; in the present context, we have no need to settle this question.

> *... the greater the distance from the centre, the greater the proportion of those who had freckle burns.*[8] *This made it difficult to explain the burns simply by radiant heat ... at least some part of the cause was something other than radiant heat.*

I should note that Wakaki nevertheless manages to satisfy himself that the story of the nuclear bombing, which was given out in military circles very early on, is indeed true overall, even though he questions it in many details.

Additional evidence to prove that the lesions could not possibly have been caused in the claimed manner will be introduced in Section 8.2. For now, we will dismiss the idea of nuclear flash burns and turn to the more interesting question of what the *real* causes of the observed burns may have been.

7.3 Fast and slow burns

If one surveys multiple eyewitness reports, a dichotomy emerges between burns that became manifest immediately after the bombing and those that developed more slowly. We will here quote one illustrative example for each. Sumiteru Taniguchi of Nagasaki [76, p. 113] suffered burns immediately:

> *The wind from the blast, coming from behind, hurled me and my bicycle to the ground ... I think two or three minutes passed before the earth stopped trembling and I heaved myself up. ... The skin of my left arm had peeled from the upper arm to the tips of my fingers and was hanging in strips. When I felt my back and buttocks, I found that the skin there had been burned to a pulp and that only the front part of the clothes I had been wearing remained.*

The burns to Taniguchi's backside were indeed extensive (see Figure 7.4 B), and he had to lie with his face down for more than a year until the wounds finally began to heal, ultimately with severe scarring and keloid formation.

An instance of delayed skin injury in a bombing victim is described by Michihiko Hachiya [47]. The writer, a head physician who had himself been injured in the Hiroshima bombing and was subsequently cared for at his own hospital, notes in his diary between August 6th and August 8th:

(6th) *I opened my eyes; Dr. Sasada was feeling my pulse. What had happened? ... I must have fainted.*

[8] I have not seen the term 'freckle burns' used anywhere else; it seems possible that 'patchy burns' might have been a more apt translation. In any case, it is clear that Wakaki's unusual term refers to some kind of irregular, discontinuous burned area.

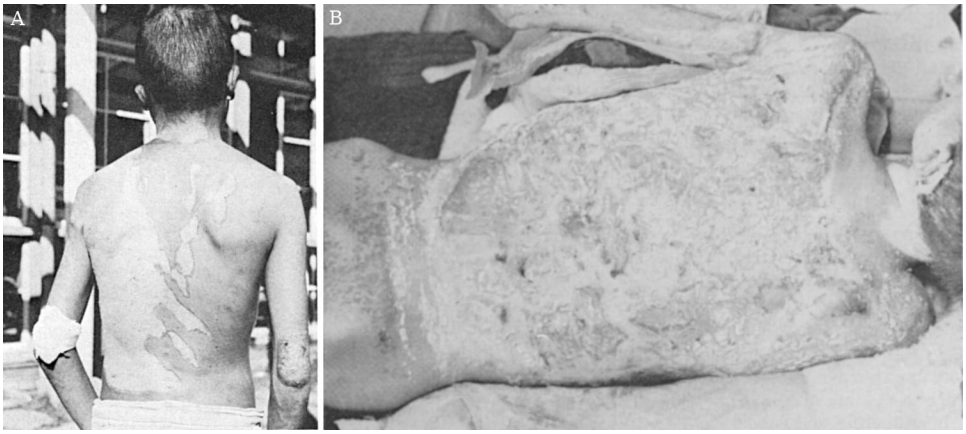


Figure 7.4 Two cases of 'nuclear flash burn' from Nagasaki. Photographs from [78], taken in October. A: This man was wearing a khaki uniform when exposed in the open at 0.5 miles from the hypocenter. (He should have died of irradiation within days.). The pattern of hypo- and hyperpigmentation on his back suggests second degree burns, perhaps due to mustard gas, while the thick scar tissue on his right elbow suggests a more severe burn, possibly by napalm. B: Extensive burns in a man 1.2 miles from the hypocenter. The details given in [78] suggest that this is Sumiteru Taniguchi (see text).

(7^{th}) *Dr. Sasada, who had looked after me yesterday, lay on my left. I had thought he escaped injury, but now I could see that he was badly burned. His arms and hands were bandaged and his childish face obscured by swelling ...*

(8^{th}) *Dr. Sasada's face was more swollen this morning than yesterday, and blood-stained pus oozed from his bandaged arms and hands. I felt a wave of pity when I thought how he had used those hands to help me two days ago.*

Further on in his diary, Hachiya reports how Dr. Sasada later develops symptoms of bone marrow suppression, but ultimately recovers. From Hachiya's description, it is apparent that Sasada's hands were injured not in the bombing itself; he could not have felt Hachiya's (presumably faint and rapid) pulse with wounded, bandaged hands. His burns sprung up only after he had tended to many victims who, like Hachiya himself, had been more severely injured outright.

Mr. Taniguchi's immediately manifest burns and tattered clothes strongly suggest that he was hit directly with some sort of incendiary, most likely napalm. In contrast, the most straightforward explanation for Dr. Sasada's delayed burns is that, by touching the skin and clothes of his patients who had been

Figure 7.5 Victims of the napalm attack at Trang Bang, South Vietnam, on June 8th 1972. A: minutes after the bombing, a girl in the nude (Kim Phuc) is running toward a group of photographers. She has severe burns, whose full extent is apparent only from behind (B). Coming to her aid in B is Nick Ut, the photographer who has just snapped the picture in A. The boy in both frames is Kim's elder brother. C: Kim's grandmother carries her grandchild Danh, Kim's cousin, who is extensively burned and will die within the hour. Scorched skin is peeling from his foot and backside.

contaminated with sulfur mustard, he was himself exposed to toxic quantities of it. His swollen face and subsequent symptoms of bone marrow suppression are likewise suggestive of mustard gas exposure.

The limited available data do not permit us to estimate the relative abundance of each type of burn; we will therefore merely discuss qualitatively the evidence which supports the assumed cause in either type.

7.4 Evidence of napalm burns

According to his description of his own travails on August 6th, Hachiya himself, like Mr. Taniguchi, was most likely burned by napalm, possibly with some additional mustard lesions as well. As he struggles towards the hospital, bereft of his clothes, he observes:

> *Others moved as though in pain, like scarecrows, their arms held out from their bodies with forearms and hands dangling. These people puzzled me until I suddenly realized that they had been burned and were holding their arms out to prevent the painful friction of raw surfaces rubbing together. A naked woman carrying a naked baby came into view. I averted my gaze. Perhaps they had been in the bath. But then I saw a naked man,*

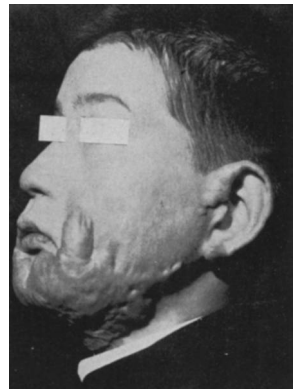


Figure 7.6 Splash burn to the face and neck caused by napalm and gasoline. This picture appears as Figure 7 in Block and Tsuzuki [133].

> *and it occurred to me that, like myself, some strange thing had deprived them of their clothes.*

Have we seen something like this anywhere else? Considering the widespread use of napalm—large amounts were dropped on Japan, and even larger ones on Korea and Vietnam—generally accessible information on napalm is astonishingly scarce.[9] However, there is one very widely known picture of a napalm victim: Kim Phuc, a Vietnamese girl who in 1972 suffered burns when her village in South Vietnam was attacked by the country's own air force (the village had been infiltrated by the Vietcong). This picture (Figure 7.5A) shows her running in the nude, in the 'scarecrow' posture also described by Hachiya. The real extent of her burns is only visible from another angle (Figure 7.5B), which also reveals the immediate peeling of the skin. Peeling and flapping skin are likewise apparent in Kim's even more severely burned cousin Danh (Figure 7.5C). While the little boy died within an hour of the attack, Kim survived. Even with expert surgical treatment, however, her burn wounds turned into extensive scars that resemble the keloids shown in Figure 7.3.

The pronounced tendency of 'nuclear flash burns' to heal with keloid formation has often been noted [136]. The same is true of napalm burns. According to the Russian physician Plaksin [131], keloid formation was observed in 52.7% of all patients in a series of 1026 Korean napalm burn patients cases. The author ascribes this to the high amount of heat transferred from the burning napalm to the adjacent tissues. While pictures of 'nuclear flash burns' abound, those of

[9] As of this writing, a simple search for "napalm" on PubMed retrieves 29 articles, of which 7 (seven) are written in English. For comparison, a search for "mustard gas" (with quotes) returns 1935 hits.

napalm burns from conventionally firebombed Japanese cities such as Tokyo are surprisingly hard to find, even though survivors with napalm burns should have been common enough. I have only found one such picture, which is shown here as Figure 7.6. According to the source [133], the victim was burned in the Tokyo firebombing when burning napalm hit a nearby fuel barrel and caused it to explode. The effect of burning gasoline on the skin would have been similar to that of burning napalm itself.[10]

In summary, the evidence strongly suggests that those of the burns in Hiroshima and Nagasaki that were manifest immediately, accompanied by burning and stripping of clothes, and followed by keloid formation, were caused by napalm. While rare, explicit accounts of exposure to napalm or a similar substance can indeed be found. John Toland [58, p. 803] relates this experience by a boy in Nagasaki:

> *Hajime Iwanaga, who would be fourteen the next day, was bathing in the Urakami River near the torpedo factory. He … exuberantly ducked his face in the water as the* pika[11] *flashed. Seconds later he emerged into a blinding world. Something warm clung to his left shoulder. It was yellowish. Mystified, he touched it and saw skin come off. He splashed toward the bank as the sky darkened ominously, and was reaching for his clothes when two dark-green spheres, the size of baseballs, streaked at him. One struck his shirt, set it afire, and disintegrated.*

Those green spheres carried fire, but did apparently not cause any harm through kinetic impact, which means that they consisted of some soft, incendiary material, much like napalm. The material on the shoulder may have been a chunk of napalm, too, that was extinguished when the boy dove underwater. For comparison, here is Kim Phuc's recollection:

> *Her first memory of the engulfing fires was the sight of flames licking her left arm, where there was an ugly, brownish-black gob. She tried to brush it off, only to scream out at the pain of the burn that had now spread to the inside of her other hand.*

[10] The senior author of this study is the very same Dr. Masao Tsuzuki who had a run-in with American censors when giving voice to the widespread perception of poison gas at Hiroshima (see Section 1.4.3). When Tsuzuki published this study on flash burns, censorship was still in force, which may have influenced his restrained commentary on the great similarity of gasoline burns and nuclear flash burns.

[11] Hachiya [47] explains the term 'pika' as follows: "*Pika* means a glitter, sparkle. or bright flash of light, like a flash of lightning. *Don* means a boom! or loud sound. … Those who remember the flash only speak of the '*pika*'; those who were far enough from the hypocenter to experience both speak of the '*pikadon*.'"

In both cases, the size and texture of the lumps of incendiary material described are consistent with those of napalm [129].

7.5 Chemical burns by mustard gas

In Section 1.4, we noted the similarity of skin lesions described by John Hersey in victims of the Hiroshima bombing to those observed by Alexander [18] in the mustard gas casualties at Bari. Eyewitness testimony from Hiroshima and Nagasaki further suggests that chemical burns to the skin by mustard gas were common. Kiyoko Sato, a girl from Hiroshima, had been evacuated to the countryside and returned to the city about a week after the bombing. Upon arrival, she finds her mother just a few moments after she has died [76, p. 55]:

> *If I had only walked a little faster, I would have been in time! I was distressed that I had not been able to see her alive and cried loudly. My mother's face was covered in blisters and had swollen to twice its normal size, and her hair had fallen out. She was unrecognizable as the mother I had known so well.*

A boy from Nagasaki, Yoshiro Yamawaki, walked across the city in search of his father on the day after the bombing, together with his twin brother [137]:

> *There were many dead bodies amongst the debris littering the roads. The faces, arms and legs of the dead had become swollen and discolored, causing them to look like black rubber dolls. As we stepped on the bodies with our shoes, the skin would come peeling off like that of an over-ripe peach, exposing the white fat underneath.*

Neither witness mentions any scorching of the dead bodies in question, and both descriptions match the known appearance of mustard gas burns.[12] We already noted above instances of burns becoming manifest only on the next day or occurring under clothing that remained intact; neither incendiaries nor flash burn can account for these observations. Having already considered the evidence that points to mustard gas as the cause of 'radiation disease', we now see that the expected skin lesions were prevalent also.

7.6 Appendix: experimental flash burns to the skin

The light intensities assumed to have been released by the bombings in Hiroshima and Nagasaki are shown in Figure 7.1 A. The surface temperature of the

[12] Strictly speaking, the skin peeling off after a mustard burn would expose not the subcutaneous fat (as stated by the boy) but rather the layer of connective tissue above it (the dermis).

'ball of light' at its most luminous stage should be in the range of 5000-7000 °K [128], which is similar to that of the sun. Therefore, the supposed atomic flash can be likened to a brief, intense pulse of sunlight, with similar proportions of ultraviolet, visible, and infrared light.

A number of experimental studies on animals and on human volunteers have attempted to model the flash burns produced by nuclear bombs. In a study on dogs [138], a thermal dose of 8 cal/cm^2 was applied to 20% of the body surface. Figure 7.1A shows that this intensity is well within the range of intensities expected near the hypocenter. Mortality was was relatively low (2 dogs out of 30) and due to septicemia. The wounds appeared different from those caused by contact burns:

> *Following a flash burn of the magnitude given in this study, an eschar is formed on the burned surface. ... This initial eschar persists throughout ... Healing of the flash wound was usually complete by four weeks with the eschar acting as a protective dressing for epithelization from deep hair follicles and wound edges.*

These results suggest that peeling of the skin might not occur in nuclear flash burns. However, blistering lesions were observed in an experimental study on human volunteers [139]. At sufficiently high doses, a superficial skin layer came off one or two days after the irradiation and left behind a red, moist wound, which does resemble the observations of peeling skin in the bombing victims. This study reports several more pertinent observations that we can compare with those made in those victims:

- A dose of 2 cal/cm^2 produced only a transient erythema, which typically subsided within half an hour. This represents a first degree burn.
- With doses of 3.9 cal/cm^2 and above, erythema of the lesion itself was immediate, and it persisted until giving way to blisters, whereas the vicinity of the lesion experienced delayed and transient erythema. Thus, any lesions of at least second degree are visible in some form immediately and throughout.
- The maximal dose given—4.8 cal/cm^2—produced at least second degree burns in all volunteers, and third degree burns in some.
- While for obvious reasons the experimental flash burns were small (1.25 cm in diameter), it nevertheless was evident that the entire light-exposed area was homogeneously burned.
- The volunteers reported immediate pain, which was described as sharp or stinging and increased with the intensity of the flash.

Clothes should offer some protection against flash burns, although it is conceivable that at very high intensity the clothing might burn up, and enough heat might be left over to damage the skin underneath. A study by Mixter [140] used an animal model (pigs) to compare the doses required to set burns in nude skin to those required with one and with two layers of fabric, respectively. With nude skin, the doses are similar to those determined in humans by Evans et al. [139]. While Mixter's data do show some scatter, a reasonable approximation is that each layer of fabric raises the energy threshold for a burn by a factor of 2.5. Thus, burns beneath two layers of fabric—which can be assumed to have been present in most victims at least around the hips—would require about 6 times more energy than on exposed skin.

8. Early clinical and pathological findings in the bombing victims

> The use of poison gas is forbidden, but wasn't this suffering worse than poison gas?
>
> Yasuko Ise, high school student from Hiroshima [75]

Chapter 5 discussed the acute medical effects of the bombings from a quantitative, statistical point of view. In this chapter, we will look at them in more qualitative detail. The evidence available for this purpose is limited. In the hours and days following the bombings, chaos reigned, and none of those who died during this time received adequate diagnosis and treatment. Their sufferings and symptoms are described only in the scattered testimony of eyewitnesses, both laypersons and medical doctors, who were anguished as much by the apocalyptic scenes around them as by their inability to help.[1]

Important sources for this chapter include the recollections by two Japanese physicians. From Hiroshima, we have the diary by Michihiko Hachiya [47], a detailed account by an experienced, perceptive, and compassionate observer, which should be read by anyone interested in the humanitarian dimension of the disaster. From Nagasaki, there is the report by Tatsuichiro Akizuki [89], a more junior physician, who unlike Hachiya was not himself incapacitated in the attack[2] and therefore was able to give more detailed observations on the victims immediately after the bombing. The reports by Oughterson and colleagues [28, 78] will again be used. The one by Oughterson and Warren [78] includes a chapter by Liebow et al. on the autopsy material collected mostly by Japanese pathologists, which was published independently as a journal article elsewhere

[1] Even though Japan had capitulated on August 15$^{\text{th}}$—9 days after the bombing of Hiroshima, and 6 days after that of Nagasaki—the U.S. did not send any physicians or medical supplies at all to either city until September, and even then gave only meager support [31]. The purely investigative Joint Commission arrived only on October 12$^{\text{th}}$ [59]. This prolonged failure to assist and to investigate seems to have been deliberate.

[2] At some later time, Akizuki did experience symptoms of ARS such as fatigue and loss of hair; admirably, however, he stayed with and cared for the patients under his watch throughout the entire time.

[38]. We will also refer to Bloom et al. [22], who describe an extensive set of experiments on the pathology caused by ionizing radiation that had been carried out in the first half of the 1940s.

In addition to the above books and reports, which were all written by physicians or medical scientists, important detail can be found in eyewitness accounts by non-specialists. The compilations of such testimony by Osada [75] and by Sekimori [76] are particularly valuable.

8.1 Clinical picture in early fatalities

8.1.1 Symptoms apparent immediately after the bombing. On the day of the Nagasaki bombing, Dr. Akizuki [89] was on duty at a hospital in the Urakami district, 1800 m from the hypocenter. The building was damaged and partly destroyed by fire, but all of the staff and the patients quickly escaped and initially survived.

The attack had occurred at 11 a.m.; shortly afterwards, the first victims from outside began to arrive, seeking help:

> *About ten minutes after the explosion, a big man, half-naked, holding his head between his hands, came into the yard towards me … 'Got hurt, sir,' he groaned; he shivered as if he were cold. 'I'm hurt.'*
>
> *I stared at him, at the strange-looking man. Then I saw it was Mr. Zenjiro Tsujiomoto, a market gardener and a friendly neighbor to me and the hospital. I wondered what had happened to the robust Zenjiro. 'What's the matter with you, Tsujimoto?' I asked him, holding him in my arms.*
>
> *'In the pumpkin field over there—getting pumpkins for the patients—got hurt…' he said, speaking brokenly and breathing feebly. It was all he could do to keep standing. Yet it didn't occur to me that he had been seriously injured.*
>
> *'Come along now,' I said, 'You are perfectly all right. I assure you. Where's your shirt? Lie down and rest somewhere where it's cool. I'll be with you in a moment.'*
>
> *His head and his face were whitish; his hair was singed. It was because his eyelashes had been scorched away that he seemed so bleary-eyed. He was half-naked because his shirt had been burned from his back in a single flash. …*
>
> *Another person who looked just like him wandered into the yard. … 'Help me,' he said, groaning, half-naked, holding his head between his hands. He sat down, exhausted. 'Water … Water …' he whispered.*

> *As time passed, more and more people in similar plight came up to the hospital—ten minutes, twenty minutes, an hour after the explosion. All were of the same appearance, sounded the same. 'I'm hurt, hurt! I'm burning! Water!' ... Half-naked or stark naked, they walked with strange, slow steps, groaning from deep inside themselves ... they looked whitish. ... One victim who managed to reach the hospital asked 'Is this a hospital?' before suddenly collapsing on the ground. ...*
>
> *'Water, water' they cried. They went instinctively down to the banks of the stream [below the hospital], because their bodies had been scorched and their throats were parched and inflamed; they were thirsty. I didn't realize then that these were the symptoms of 'flash burn.'*

Thus far, Akizuki has described victims whom he had encountered within one hour or so of the attack. At this early stage, we can make the following observations:

- Akizuki does not immediately recognize his 'strange-looking' neighbor, which suggests that his features are already somewhat distorted. They will be much more so later in the day.
- He notices some signs of immediate burns—singed hair and eyelashes, as well as nudity (see Section 7.4).
- Akizuki does not describe any other outward signs of injury; instead, he reassures his suffering neighbor that he is alright.
- The victims speak hoarsely; their throats are 'parched and inflamed,' and they are thirsty; their breath is labored.
- The victims are pale and weak, and some collapse.
- The victims are holding their heads between their hands, suggesting that they have a severe headache.

8.1.2 Symptoms apparent after several hours. While the above observations capture the early stage of the injuries, the victims' aspect is strikingly transformed later on. Here is Akizuki's description:

> *In the afternoon a change was noticeable in the appearance of the injured people who came up to the hospital. The crowd of ghosts which had looked whitish in the morning were now burned black. Their hair was burnt; their skin, which was charred and blackened, blistered and peeled. Such were those who now came toiling up to the hospital yard and fell there weakly.*

These victims might have come from another district of the city, further away from the hospital but closer to the hypocenter, where they might have suffered more severe immediate burns. However, a similar change is also apparent in the victims who had arrived earlier. While on his way to help an injured colleague, he again encounters some of them:

> *When I reached the little river, I came across an astonishing scene. Half-naked or nearly naked people were crouching at the water's edge. All looked alike, without distinction of sex or age; long hair was the only clue to the female sex. On one side their bodies had been grilled and were highly inflamed. The procession of white ghosts which had passed me some time before had gathered here on the bank of the stream, seeking water to relieve the terrible thirst and the scorching pain of their bodies. Crowds of these victims lined the stream.*
>
> *'Oh, how it hurts! I'm hurting—burning!' said Mr. Tsujimoto, groaning. His face, which had been whitish, when I saw him earlier, was now darker, blackened; his lips were swollen. His wife sat not far away, her face and body also blackened, moaning insensibly.*

It is clear that, in this group of patients at least, overt symptoms have become manifest with a delay of several hours.[3] They are now obvious even in the wife of Mr. Tsujimoto, whom Akizuki had not even mentioned as being afflicted earlier on. Still later in the day, Akizuki describes both Mr. and Mrs. Tsujimoto as 'cinder-burnt.' While Mrs. Tsujiomoto will live for a few more days, her husband expires the same night:

> *At about midnight, Mr Tsujimoto's condition suddenly worsened. … By degrees, Mr. Tsujimoto's breathing became harsher. I couldn't feel any pulse. … Suddenly Mr. Tsujimoto went into a violent fit of convulsions; his eyes bulged. 'His last moment has come!' said someone.*

Labored breathing in the bombing victims is confirmed by another eyewitnesses from Nagasaki, Akira Nagasaka [76, p. 74]:

> *A woman, probably in her mid-thirties, was lying on the ground, her hair wild, her clothes in tatters, her face red with blood. She was putting all*

[3] While Akizuki's statement that 'on one side their bodies had been grilled' might suggest some sort of real flash burn, he later also notes that some of the patients had burned faces and backs, for which he gamely asserts some contortionist explanation. This echoes Oughterson and Warren [78], who twist the protagonists of their case descriptions into the most unnatural poses for the same reason.

the strength that remained in her to raise her head and murmur, "Water, water."

When I had gathered my wits about me, I scooped some dirty water out of a nearby ditch and gave it to her. She drank it as if it were the most delicious thing ever to pass her lips, but most of it merely trickled down her chin onto her breast. "More, please," she begged, but she could do no more than gasp for breath when I brought it, having no strength left to drink.

The testimony from Hiroshima is, if anything, even more gruesome.[4] Eyewitness Kosaku Okabe [76, p. 35] was not near the hypocenter for the bombing, but he came upon the scene in downtown Hiroshima several hours afterwards:

Wherever a puddle of water had collected from burst water pipes, people had gathered like ants around a honey pot. Many had died where they lay at the water's edge, their strength gone. Others had clambered over the dead bodies to get at the water, only to die in the same way, their bodies piling one on top of another.

Okabe also describes the aspect of the victims:

Most people had been wearing light summer shirts that morning. But most of the dead were bare chested, and many were completely naked, perhaps because their clothes had been burned off them. The parts of the body that had been exposed to the flash had suffered great burns, and the skin was turning purple and trailing from the body in strips.

In every case, the eyeballs of the dead were either protruding from their sockets or hanging out completely. Blood had gushed from the mouth, ears, and nose. The tongue had swelled to the size of a golf ball and had pushed its way out of the mouth, gripped tightly by the teeth. The whole anatomy seemed to have been destroyed. Most bodies were bloated, and it was often impossible to tell whether they were male or female.

The grisly, apocalyptic picture painted by Okabe's testimony might seem exaggerated, but each detail is confirmed by other eyewitnesses [75, 76, 141]. While the victims described by Okabe are already dead, another witness depicts the scene when some of them were still alive. Hachiya [47] relates the observa-

[4] We noted earlier that the incidence of both radiation sickness and burns in victims near the hypocenter was lower in Nagasaki than in Hiroshima, even though the bomb yield in Nagasaki is said to have been higher (see Chapters 5 and 7). It seems likely that less mustard gas, and perhaps also less napalm, was used in the second bombing than in the first.

tions told him on August 6th by one of his visitors, a Mr. Hashimoto, who like Okabe entered the inner city after the bombing:

> *When I reached the Misasa railway bridge ... I encountered a dead man. I saw many others in the water tanks fighting for breath. The sight was horrible.*

Mr. Hashimoto also describes the days following the bombing:

> *During those days, wherever you went, there were so many dead lying around it was impossible to walk without encountering them—swollen, discolored bodies with froth oozing from their noses and mouths.*

Overall, the testimony given by several independent witnesses from both cities is remarkably consistent. We therefore can't dismiss it, but instead must try to understand what exactly could have caused such terrible injury and disfigurement.

8.1.3 Pathophysiological interpretation of early symptoms. Before identifying the causes, we must take a step back and consider what the clinical signs observed in these victims tell us about the underlying pathophysiology.

Skin burns. A key observation here is that in some victims at least, such as the Tsujimotos, burns of the skin were manifest only after some hours, as is typical with mustard gas. The blackened aspect of the skin in such cases was most likely caused by intense cyanosis rather than 'scorching,' which should have been apparent immediately (see for example Figure 7.5). It is quite likely, of course, that some victims suffered both immediate (napalm) and delayed burns.

Circulatory shock and capillary leak syndrome. The initial paleness reported by Akizuki in patients who arrived on foot at his clinic suggests beginning circulatory shock. At a more advanced stage of shock, paleness may give way to cyanosis; this is observed by Akizuki in some of the initially pale patients at a later time, and it is also described by Okabe in the victims that he encounters several hours after the Hiroshima bombing.

Shock may be accompanied by capillary leak syndrome, which causes intense thirst and, after intake of large volumes of water, extreme edema (Figure 8.1). All of these symptoms were described in the bombing victims.

A related observation is the acute headache, which is suggested by Akizuki's description of patients holding their heads between their hands. Headaches are caused by vascular distension in the meninges; the simultaneous occurrence with shock suggest that the latter is likely caused in part by the loss of vascular tone.

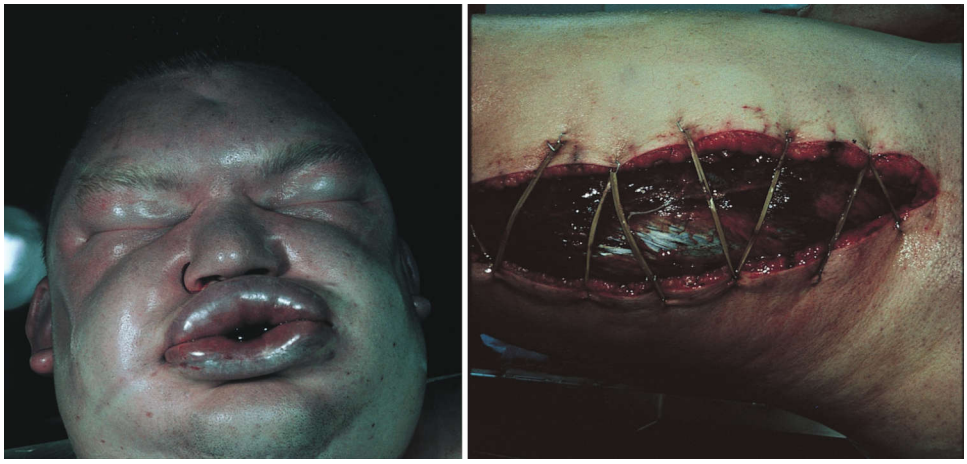


Figure 8.1 Patient with capillary leak syndrome (deceased; [142]). Left: the face is cyanotic and extremely swollen. Right: swelling of a limb has led to fascial compartment syndrome, in which nerves and blood vessels are compressed by the edema within a tightly confined space. Transient incision of the fascia (a sheet of firm connective tissue) that encloses the compartment was carried out to relieve the compression.

Injury to the lungs and airways. Acute respiratory distress is described in early fatalities, but both Hachiya and Akizuki also report labored breathing in the patients they examine in the subsequent days and weeks. Immediate affliction of the upper airways can be surmised from Akizuki's observation of hoarseness in the victims he meets shortly after the bombing.

The froth oozing from mouths and noses noted by the sharp-eyed Mr. Hashimoto in the dead bombing victims indicates severe pulmonary edema, while outright bleeding from the mouth and nose, described by Okabe, suggests injury to blood vessels in the mucous membranes of the airways, and possibly to larger vessels inside the chest.

Also pertinent is Dr. Masao Tsuzuki's remark on the 'suffocating pain' experienced by those who inhaled the gas which 'permeated immediately after the explosion of the atomic bomb' (see Section 1.4). Overall, it is plain that some noxious agent released at Hiroshima and Nagasaki attacked the lungs and airways.

Traumatic asphyxia and orbital compartment syndrome. With the assumptions of capillary leak syndrome and injury to the lungs and airways, we can account for the thirst, the general edema, the respiratory distress, the cyanosis, as well as the blood and froth spilling from the mouth and nose. The peeling skin is, at this point of the exposition, no longer a mystery. That leaves the

bleeding from the ears and the eyeballs protruding or even hanging out. How can we fit these into the picture?

The protruding eyeballs are a telltale sign of *orbital compartment syndrome*. The eye socket (Latin: *orbita*) is a confined space, and if some irregular process such as edema or hemorrhage claims some of that space, then the eyeball is displaced outwards. One contributing factor would have been the capillary leak syndrome, but there most likely was another one—*traumatic asphyxia*, also known as *Perthes syndrome*. Most commonly, traumatic asphyxia is triggered by compression of the thorax, but it can also occur with other causes of disrupted respiration, including severe asthma attacks [143]. It arises when pressure to the chest or injury to the lungs prevents blood pumped by the right heart from entering the lungs. The blood therefore backs up in the right heart and into the large veins that supply it, particularly those within the head. Blood vessels become distended, the blood stagnating within them becomes desaturated of oxygen, its color turning dark, and plasma fluid leaks into the tissues; the patient's face turns purple and swollen. Bursting blood vessels may cause bleeding from all cranial orifices, including the ears. Bleeding could likewise have occurred behind the eyeballs; in fact, the convulsions and bulging eyeballs in the dying Mr. Tsujimoto suggest some such event. Since severe lung damage was present in the bombing victims, we can conclude that the preconditions for traumatic asphyxia were met.

While traumatic asphyxia and ocular compartment syndrome are both rare in normal life, there is indeed a clinical case report that describes them in combination [144]. As it turns out, the severely injured patient in this case also developed capillary leak syndrome. The authors state that capillary leakage preceded the orbital compartment syndrome, and they consider it a contributing cause of the latter.[5]

Based on the foregoing, it stands to reason that the combination of lung and vascular injury present in the bombing victims could also account for the development of orbital compartment syndrome. We can thus reduce the overall clinical picture to three fundamental pathophysiological effects:

1. injury to the lungs and airways;
2. injury to the vasculature, leading to capillary leak syndrome and shock;
3. injury to the skin, causing it to peel.

[5] According to Fred and Chandler [143] and Dwek [145], lasting ocular injury, suggestive of damage by increased pressure within the eye sockets, is common in traumatic asphyxia even without manifest capillary leak syndrome. Dwek explains exophthalmia (protruding eyeballs) in such patients with hematoma in the eye socket, but with the limited diagnostic means available in his day, distinguishing hematoma from edema behind the eyeball would have been difficult.

8.1.4 Causal attribution. What could have caused these three effects? The easy part of the answer is that neither 'flash burn' nor ionizing radiation can account for this entire clinical picture. As discussed in Section 7.6, flash burns should have been visible in some form immediately, but Akizuki fails to notice them in several patients whom he encounters shortly after the bombing. Without very severe exterior burns, there simply is no mechanism by which a flash of light could produce acute respiratory distress.

As regards ionizing radiation, here is the case description of a patient who received approximately five times a lethal dose of it [71, p. 218]:

> *In a nuclear criticality accident at Los Alamos in 1958, one worker received a total body dose of mixed neutron and γ-radiation estimated to be between 39 and 49 Gy. Parts of his body may have received as much as 120 Gy. This person went into a state of shock immediately and was unconscious within a few minutes. After 8 hours, no lymphocytes were found in the circulating blood, and there was virtually a complete urinary shutdown despite the administration of large amounts of fluids. The patient died 35 hours after the accident.*

This patient received a dose of radiation about as high as it could have been near the hypocenter in Hiroshima. He promptly developed cerebrovascular syndrome and also general circulatory shock, and he quickly died of it—without intensive care, he probably would have died on the same day, as did many of the victims in Hiroshima and Nagasaki. However, no mention is made of facial or general cyanosis, respiratory distress, peeling skin, or dangling eyes. Since he lost consciousness so quickly, he would not have had time enough to find a puddle and drink enough water to swell up to any great extent. Thus, apart from shock and rapid death, his clinical picture bears no resemblance to that described in the victims at Hiroshima and Nagasaki.

Animal experiments reported by Bloom et al. [22] showed the lungs to have relatively low susceptibility to radiation; lethal doses of X-rays or neutrons produced little or no evidence of tissue damage when compared to controls. The skin, too, showed very minor effects at such doses. While these findings do of course not rule out lung or skin damage with supra-lethal irradiation, they exclude *preferential* damage to these organs, which is evident in the Hiroshima and Nagasaki victims.[6]

[6] While Bloom [22] was published only in 1948, the experiments described were carried out mostly before 1945. From the great variety of radionuclides they used, it is clear that Bloom and colleagues must have had high-priority access to novel isotopes as these became available through ongoing research in Fermi's laboratory. Considering that the reports by Bloom and

The more difficult and interesting part of the answer concerns how we actually *can* account for the clinical picture. Since we already have evidence that napalm and mustard gas were used, we will examine if they can explain it.

Napalm. Mr. Tsujimoto, the patient most thoroughly described by Akizuki (Section 8.1.1), has lost his shirt in the bombing, and his hair and eyelashes are singed. Even though he does not present any obvious burns at the time, this does suggest some possible exposure to napalm, albeit probably not through a major direct hit.

According to Björnerstedt et al. [129], the fire from a sufficiently large napalm bomb will inflict harm through radiating heat even at some distance. Moreover, conventional burns can cause smoke inhalation injury, which can result in acute respiratory distress with rapid deadly outcome [146]. Severe burns will also cause circulatory shock; and with napalm, this may occur even when only some 10% of the total body surface have been burned [147]. Thus, napalm could in principle set off the pathophysiological cascade that would produce all of the symptoms seen in the early fatalities, and this may well have happened in some of them.

It is doubtful, however, that napalm was the only cause in Mr. Tsujimoto's case. Smoke inhalation injury tends to occur with fires indoors, since here the smoke accumulates in a confined space; Mr. Tsujimoto, however, reported having been hurt while harvesting pumpkins in the field. Conceivably, one might also suffer smoke inhalation injury outdoors, if surrounded and trapped by fire; but it seems unlikely that one could escape such an inferno without also receiving significant burns to the skin. According to Dolinin [147], asphyxia occurs in approximately 5% of napalm victims, particularly in those with manifest burns to the face. Overall, napalm seems unlikely as the cause of respiratory distress in Mr. Tsujimoto, or in the other victims with similar early symptoms. We thus should consider the possible role of mustard gas.

Mustard gas. I should note upfront that the literature does not report any clinical cases of mustard gas poisoning which exhibit the complete picture described in Section 8.1.2. While capillary leak syndrome and extensive damage to the skin and lungs are documented (see Chapter 6), I have not found a

by Oughterson and Warren [78] were both prepared under the auspices of the Atomic Energy Commission, it is peculiar that Oughterson and Shields do not cite Bloom. Even more bizarrely, Bloom's 800 page volume—at least within its otherwise very extensive index—does not even mention the atomic bombings. Thus, no connection is ever made between Bloom's experimental work and the clinical or pathological observations in Hiroshima and Nagasaki. Bloom does briefly note that in some experiments mustard gas was tested in parallel with radiation but gives no details on the outcomes.

single case report on traumatic asphyxia caused by mustard gas. Nevertheless, I propose that exactly this did occur at Hiroshima and Nagasaki. My reason is that the bombing victims must have sustained much graver acute lung injury than any earlier victims—their sufferings, even though caused by poison gas, were indeed 'worse than poison gas'. In World War I, mustard gas was introduced only after other poison gases had been, so that the soldiers who encountered it were already equipped with gas masks. Likewise, gas masks had also been worn by the poisoned mustard gas factory workers described by Warthin and Weller [98]. In contrast, the victims at Hiroshima and Nagasaki had no warning and no protection, and they must have inhaled the gas in far greater amounts than those earlier victims.[7]

As a consequence of such high doses to the lungs, the airways would have become clogged by swelling mucous membranes, fibrin plugs, and blood clots (see Section 6.3.2). Air becoming trapped behind such obstacles—acute emphysema—would have raised the pressure inside the chest and compressed the pulmonary veins, thus preventing the flow of blood returning from the body and the head. Additionally, clots would have formed within the lungs' blood vessels themselves, further impeding the flow of blood back into the lungs. In the most severely poisoned victims, the acute obstruction of the airways and the lung circulation would have been incomparably worse than in any asthma attack.

Pulmonary effects similar to those just described for mustard gas have also been documented for smoke inhalation injury [149], which is common in napalm burn victims.

8.1.5 Conclusion. In summary, therefore, I propose that both napalm and mustard gas can account for the full clinical picture observed in the early fatalities, while radiation cannot. Mustard gas was very likely the dominant cause in those victims who initially appeared to be free of burns, such as Mr. Tsujimoto, but napalm may well have contributed significantly in many other victims.[8]

[7] The most similar scenario may have occurred in Iranian soldiers subject to Iraqi mustard gas attacks. Freitag et al. [148], who report on some Iranian veterans with severe chronic bronchopulmonary damage, also state that "many soldiers died immediately on the battle field, probably due to acute chemical-induced pulmonary edema." The surviving victims reported that "they first noticed a bitter taste and a garlic-like smell immediately after the exposure to the poison gas. Minutes to hours later, dizziness, headaches, and shortness of breath were common complaints." The authors raise the possibility that lung poisons other than sulfur mustard may have been used, but I have not found this corroborated in other sources.

[8] While I see no need to postulate the use of other lung poisons, it can of course not be ruled out. Both chlorine and phosgene were used in World War 1 and caused acute and severe lung damage [98, 126]. Another plausible candidate is lewisite, which is known to have been produced and studied by the U.S. during World War II [17], and whose acute effects resemble those of

8.2 Acute retinal burns: the dog that didn't bark

When exposed to a nuclear detonation, the eyes may be harmed both by the flash of light and by ionizing radiation. The latter most commonly causes *cataract*, that is, increased opacity of the lens, which typically becomes manifest with a delay of several months or years. An increased incidence of cataract has indeed been repeatedly described in survivors from Hiroshima and Nagasaki; this will be considered elsewhere xref. Here, we will focus on the acute lesions that were observed very shortly after the bombing, as well as those that were not observed but should have been.

We have seen earlier that most of the skin burns observed in Hiroshima and Nagasaki were ascribed to the flash of light from the detonation. This raises the question how the same flash would have affected the eyes. The intuitive expectation is that it should have significantly harmed them. Dr. Oughterson thought so, too, according to the ophthalmologist John Flick [150]:

> *"They say this explosion gives off the light of ten-thousand suns!" he [Oughterson] said to me. "If this be true there should be something for you to do."*

While the 'ten-thousand suns' estimate is as vague as it is dramatic—to give it a specific meaning, the distance of measurement would also be required—the question of ocular lesions by nuclear detonations has been investigated both in humans and animals.

8.2.1 Retinal burns observed in humans after later bomb tests. Probably everyone has been warned against looking at a solar eclipse with unprotected eyes. Doing so may cause circumscribed burns on the retina, which will leave behind a permanent defect in the field of vision (a *scotoma*). The same would be expected in people who happen to look at a nuclear flash, and indeed Rose et al. [151] have reported on six American soldiers who developed just such burns after looking at the fireballs of later nuclear tests from distances up to ten miles. The authors also explain why retinal burns may occur at such large distances from the detonations; the reason is illustrated in Figure 8.2. While the light intensity at the pupil decreases with the square of the distance, this effect is exactly compensated by the diminishing size of the retinal image. The

sulfur mustard but arise more rapidly, probably due to its greater volatility [32]. The reasons why lewisite is unlikely to have been used *instead* of mustard gas are discussed in Chapter 6; the reasons given there apply to the other two poisons also.

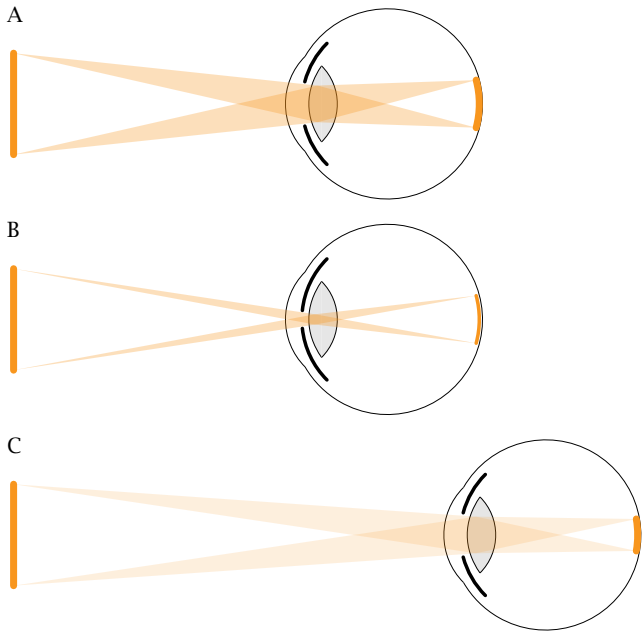


Figure 8.2 Effects of pupil diameter and of object distance on retinal image. A: All light that originates from the same point on the object and falls onto the aperture (pupil) is focused onto the same point on the retina; this creates an inverted image of the object. B: If the pupil narrows, the size of the retinal image remains unchanged, but its intensity is reduced. C: If, relative to A, the pupil diameter stays the same but the object distance increases, then the light that falls onto the pupil is 'diluted', but this is compensated by the reduced size of the image—the intensity of the retinal image stays the same.

brightness of the latter decreases only in proportion to the haziness of the air, which thus becomes the limiting factor.[9]

The size of the pupil also limits the light intensity at the retina, of course; that is, after all, its purpose. Since the pupil is wider at night than during the day, it follows that retinal burns will occur at greater distances by night. Rose et al. [151] do not provide any details on the time of day or the magnitude of the detonations that occasioned their clinical cases, which means that we cannot directly apply their findings to the conditions at Hiroshima and Nagasaki.

[9] Another limitation would be the less than perfect optical precision of the eye's refractive elements (cornea and lens), but within a few kilometers from the detonation this should not matter much.

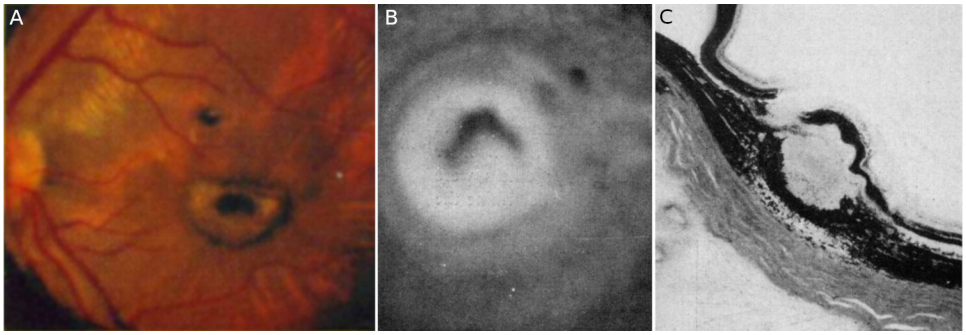


Figure 8.3 Nuclear flash burns of the retina in a human and in a rabbit. A: retinal burn in a soldier exposed 2 miles from the detonation, photographed 6 weeks after the event [151]. B: Early stage of retinal lesion in rabbit. C: Histological section through rabbit retinal lesion. The band of gray tissue is the sclera; the dark layer comprises the choroid and the retina. The retina is bulged and ruptured. B and C from Byrnes et al. [152].

The quantitative aspects of retinal burns are somewhat more explicitly addressed by Byrnes et al. [152]. These authors present studies on 700 rabbits, which were exposed to the flashes of nuclear detonations at night, at distances of up to 42 miles. At all distances, the retinas suffered discrete burns, which decreased with distance in size and in the degree of tissue destruction. Within eight miles of the detonations, the authors describe a 'volcano-like' appearance of the lesions, with prominent edges and a deep central hole, the bottom of which they presumptively identified as the *sclera*, that is, the eyeball's sturdy outer layer of connective tissue. The rabbit eye lesions appear very similar to those in Rose's human patients (Figure 8.3).[10]

Byrnes et al. [152] do not state the magnitudes of the detonations that burned those rabbit retinas. They do, however, apply the findings from their rabbit studies to provide explicit estimates for the range at which a 'typical' 20 kT fission bomb—as described theoretically in Glasstone [128], and as purportedly used in Hiroshima and Nagasaki—should cause retinal burns in humans, by day or by night, and under various conditions of visibility. They conclude that the range would be up 40 miles by night, and some 10-20% less by day. However, they do no spell out all of the assumptions that went into these estimates, and it is not clear to me why the difference in range between day and night would be so small. Their assumed decrease of the pupil aperture from 8 mm by night to 4 mm by day will reduce the energy reaching the retina by a factor of 4; according

[10]The volcano-like appearance agrees with the mechanism of injury proposed by Byrnes et al. [152] and Vos [153], namely, a local steam explosion within the retina, caused by the very rapid absorption of energy, which allows no time for heat dissipation.

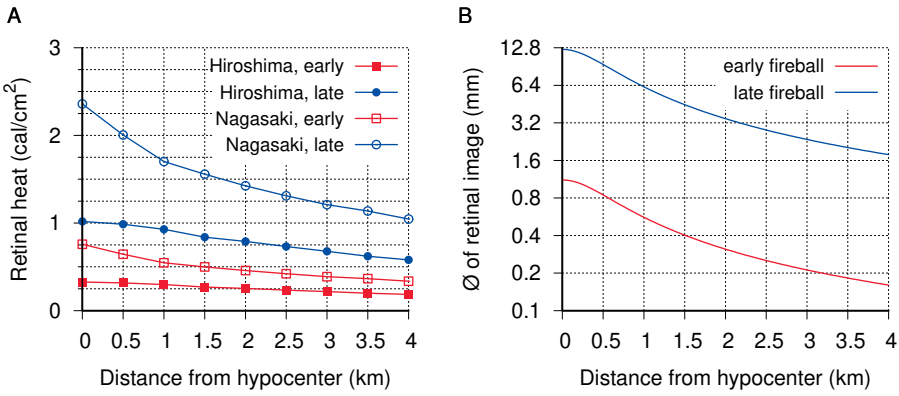


Figure 8.4 Thermal energy density (A) and diameter (B) of retinal images of the Hiroshima and Nagasaki nuclear bombs. 'Early' and 'late' in A refer to the stage of the fireball. See text for details.

to my own calculations, this should reduce the range by day to approximately half that by night, giving a maximum range a bit below the atmospheric visibility. Of note, the largest distance among Rose's case reports [151] is 10 miles.

8.2.2 Retinal doses of thermal radiation at Hiroshima and Nagasaki. To gain a firmer footing, we can estimate the heat dose to the retina at Hiroshima and Nagasaki from the thermal radiation which purportedly prevailed on the outside (see Figure 7.1A), the geometrical constraints of ocular vision (see Figure 8.2), and the transmittance of the translucent parts of the eyeball. Following Byrnes et al. [152], the latter was taken to be 0.4. The pupil diameter was fixed at 2 mm, which corresponds to full adaptation to bright sunlight—the bombings occurred on bright, sunny summer mornings—and the distance between the pupil and the retina was assumed to be 24 mm.

According to Glasstone [128], the fireball has two distinct stages of high luminosity. The 'early fireball' exists at 1 ms after the detonation. It only lasts a very short time, during which only a comparatively small cumulative amount of radiation can be released; however, its small diameter of only 27 m means that this amount will be focused onto a small retinal image, where the intensity may still reach harmful levels. The late fireball is larger (300 m across) and also much longer-lived—up to 3 seconds, but most of the energy is released within the first second. It thus reaches a higher energy density across a larger retinal image.

The results of the calculation are depicted in Figure 8.4. For interpreting them, with need to know the thermal energy which, if transferred to the retina as a very brief flash, will produce a retinal burn. Byrnes et al. [152] estimated this

value to be 0.1 $\mathrm{cal/cm^2}$, and they also state that in a separate series of experiments, which is not described in detail in the cited study (and which I have not found published elsewhere), burns were indeed induced with an only slightly higher energy (0.14 $\mathrm{cal/cm^2}$). All data points in Figure 8.4A exceed that threshold.

What are the roles of early and late fireball, respectively, in the generation of retinal burns? On the short time scale of the early fireball (1 ms), no protective lid reflex will be triggered, so that anyone with the flash in their field of vision will receive at least this dose of energy in full. On the other hand, the longer duration of the late fireball means that some of the energy may be shut out by lid reflexes. The question therefore arises to what extent the late fireball contributes to the formation of retinal burns. The sizes of the burns observed by Rose et al. [151], when compared to the predicted ones in Figure 8.4B, suggest that the late fireball does contribute significantly; but since those authors do not tell us how similar those nuclear detonations were in size to the 'typical' 20 kT bomb—if they were larger, maybe so were the early fireballs—we cannot be quite sure. In any event, even in the most stringent scenario—pupils adapted to a bright sky before the flash, and considering the early fireball only—the retinal doses of thermal radiation still exceed the burn threshold, even if only slightly in Hiroshima at some distance from the hypocenter. Overall, both theoretical considerations and previous evidence [151, 152] indicate that retinal burns should have been very common in both Hiroshima and Nagasaki.

8.2.3 Flick's eye exams in bombing victims. The ophthalmologist John Flick arrived in Japan in early September and spent several weeks examining a large number of patients in both Hiroshima and Nagasaki.[11] His report [150] is the most comprehensive and detailed of its kind. He writes:

> *At the end of the second day I had examined approximately 300 patients. I had found the usual traumatic lesions one sees in wartime but none of the corneal or lenticular syndromes I had expected to find. There were few ophthalmias among the sick and those found were of the nonspecific kind due to infection. Knowing the high degree of radioresistance of the tissues of the posterior segment I had paid little attention to ophthalmoscopic studies.*

[11] Flick notes, that on arrival, "we learned that the death rate was 100 per day among those survivors and felt that any studies made would have to be instituted quickly." This must also have occurred to other medical officers; nevertheless, Oughterson and Shield's 'Joint Commission' arrived only a full month later in October. Liebow [59] suggests that this was due to problems with weather and logistics, but these did not stop Flick, nor several other advance teams with non-medical tasks such as, it would seem, painting 'atomic bomb shadows'.

The posterior segment of the eyeball includes the retina, and its examination uses an ophthalmoscope. Thus, Flick's remark suggests that he was initially focused on the effects of ionizing radiation more than on those of the flash of light.[12] Nevertheless, shortly after, he does make a thorough study of the retinal symptoms in survivors. This is prompted by his observation of retinal bleeding in two patients with hematopoetic syndrome (see Section 5.2.1):

> *On the third day I was examining two moribund Japanese soldiers with bloody diarrhea, bleeding from the gums, covered from head to foot with petechiae. Their white [blood cell] counts were 2,000 and 900. I examined their eyegrounds. Both had extensive hemorrhagic and exudative lesions of the retina. It seemed entirely consistent with the rest of the picture … these characteristic fundus [retinal] lesions were one of the most reliable criteria of radiation sickness.*

In his paper, Flick individually summarizes and also tabulates several dozen of his cases. Of the retinal lesions he describes, he attributes not a single one to 'flash burn', nor do any of the lesions shown as illustrations exhibit the striking volcano crater aspect evident in Figure 8.3.

The dearth of clinical cases of retinal flash burn in Hiroshima and Nagasaki is acknowledged by Rose et al. [151] and Byrnes et al. [152]. Both papers do, however, cite one report which purportedly describes one actual case. From Rose et al.:

> *The literature reveals no report of such a burn except for a single case of bilateral central scotoma incurred in the Hiroshima atomic explosion.*

The clinical picture described in the reference given by Rose and by Byrnes, however—Oyama and Sasaki [154]—is not at all characteristic.[13] Thus, the medical literature does not substantiate a single case of retinal flash burns in Hiroshima or Nagasaki.

[12] The cornea has comparatively low susceptibility to ionizing radiation, and lenticular lesions tend to become manifest with delay; it is therefore not clear to me why Flick was initially concentrating on these.

[13] Both Rose and Byrnes cite this reference second-hand ('cited in Cogan …') and apparently never read it. I obtained the Japanese original and had it translated by a native speaker. It is not a full clinical case report, but only a poster abstract one page long. In translation, its title reads *A case of corneal burns by the atomic bomb*. The text describes a patient who suffered burns to the face (probably by napalm), followed by scars as well as corneal lesions; only a single concluding sentence notes that *degenerative* retinal lesions—not burns—'were also seen.' While the visual deficit (scotoma) in a true retinal burn should have been manifest immediately, it was noted by this patient only with some delay, suggesting that it arose from the scarring of the corneas; this is a well-known late effect of facial napalm burns [147].

8.2.4 Pathological findings in the eyes of deceased victims. Flick shows some histopathological pictures of retinas from deceased patients, which exhibit the sequelae of hemorrhages but again have no similarity with flash burn lesions [150]. Likewise, Liebow, who surveys the autopsy materials he had confiscated from Japanese pathologists while serving on the Joint Commission, mentions hemorrhage as the only retinal lesion [38].

Schlaegel reports his findings on autopsy materials from a series of patients at Nagasaki who had died from 'radiation sickness' approximately four weeks after the bombings [155]. He finds a variety of lesions, mostly to the anterior eye (see Section 8.3); however, he does not describe or discuss any cases of retinal flash burn. The same is true of another, shorter report by Wilder [156]. Overall, I have found not a single study that provided any evidence of retinal burns in autopsy materials from Hiroshima or Nagasaki.

8.2.5 Anecdotal reports of retinal flash burns. In contrast to the medical literature, both Akizuki and Hachiya suggest that some sort of retinal burns indeed occurred. In early September, Akizuki is visited in his hospital by an American military physician, who proceeds to examine the eyes of his patients [89, p. 131]:

> *He seemed to be an eye specialist, for he began eventually to examine the patients eyes with an ophthalmoscope … The American remarked: "Most of them have had the optic nerves of their retinas damaged by the A-bomb's flash, and their eyesight has been impaired. They may even lose it altogether."*

Similarly, in his diary entry from August 23rd, Hachiya recounts a conversation with his hospital's ophthalmologist, Dr. Koyama:

> *I asked Dr. Koyama what his findings had been in patients with eye injuries. "Those who were watching the plane had their eye grounds burned," he replied. "The flash of light apparently went through the pupils and left them with a blind area in the central portion of their visual fields. "Most of the eye-ground burns are third degree, so cure is impossible."*

On the next day, Hachiya muses about his own condition:

> *I recalled Dr. Koyama's account of patients who had been blinded by looking directly at the* pika*. Their blindness was understandable because their eye nerves had been scorched. My exposure was indirect. I had seen only the flash, but the heat rays had not reached me so the "mirrors" in my eyes were not injured.*

Hachiya's distinction between exposure to the flash and the thermal rays is fictitious, however—both visible light ('the flash') and infrared light ('thermal rays') contribute to the burns, but visible light accounts for the greater share; and both travel in a straight line—one cannot suffer one but be spared the other.

It is noteworthy that Oyama and Sasaki published their poster abstract [154] while employed in the same hospital as Hachiya and Koyama. Presumably, the authors would have had access to the patients examined by Koyama, or at least to their files. In this hospital, a significant number of autopsies were carried out in the weeks after the bombing by Okayama medical school pathologist Dr. Tamagawa. His autopsy samples were later appropriated by Liebow, who makes no mention of retinal burns (see Section 8.2.4).

That neither clinical files nor autopsies from Koyama's own hospital furnished more than Oyama and Sasaki's single case, which morphed into a 'retinal flash burn' only in the skillful hands of later American authors, strongly suggests that Koyama's diagnosis was premature. The cases he observed may have been similar to those which Flick attributed to thrombocytopenia rather than to flash burns, and which would indeed have healed in those patients who survived their ARS in the end. In my view, therefore, the anecdotal reports are lacking in substance and cannot stand up to the uniformly negative evidence from the medical literature; they are discussed here only for completeness' sake.

8.3 Other acute eye lesions

Many witnesses describe a 'blinding flash', but do not report having been unable to see afterwards. A very bright flash that stays below the burn threshold can indeed transiently suppress our vision; many will have experienced this when exposed to a photographer's flash.[14] In bright daylight, this effect will last a few minutes at most; however, some victims appear to have been blinded for longer periods of time. On August 7th, Hachiya notes in his diary:

> *I heard footsteps, and a man appeared at the door, outlined in the flickering darkness. His elbows were out and his hands down, like the burned people I had seen on my way to the hospital. As he came nearer, I could see his face—or what had been his face because this face had been melted away by the fire. The man was blind and had lost his way.*

Like the case described by Oyama and Sasaki [154] (see page 116), this one may have been caused by napalm, but the loss of vision is more acute. Hachiya

[14] If you have not, you can experience it second hand by watching Hitchcock's famous movie *Rear Window*, in which James Stewart's character, a wheelchair-bound photographer, tries without success to hold off an attacker by repeatedly blinding him with camera flashes.

does not report on the subsequent clinical course in this case. Likewise, he reports once only on another one:

> *"Has he been fed?" I asked Miss Kado. "Don't worry, Doctor," replied Miss Kado. "There are plenty of potato leaves in the garden, so I don't think he'll be hungry."*
>
> *The patient we were talking about was a horse who had been burned and blinded by the fire. Whoever saw him first did not have the heart to turn him away, so he was put in the garden under our window.*

Flick [150] describes a single case of transient blindness which lasted for several days, and which was followed by symptoms suggestive of moderately severe 'radiation sickness':

> *Furuta, a young Nagasaki woman, aged 18 years, was in Ohashi in a wooden house. She states that at the time of the explosion she was blinded and could not see for three days. From August 15th to 18th she had fever up to 40°C. At this time the cuts she had began to be infected. Fever recurred, September 4th to 14th, up to 40° C, and there was soreness of the gums and tonsillitis.*

The combination of symptoms in this case strongly suggests a causation by sulfur mustard (see Section 6.3.3 and 6.3.6). More severe exposure of the eyes to mustard gas can result in the loss of the epithelial cell layers which cover the cornea [98, p. 97]. A similar lesion was described by Schlaegel [155] in one deceased Nagasaki victim (see Figure 8.5). Schlaegel himself ascribes it to ultraviolet rays; however, if UV rays from the flash had indeed been to blame, then the concomitant and much more intense visible light should have caused severe retinal burns as well. Schlaegel also summarizes some clinical observations, related to him by Japanese colleagues, which are entirely consistent with the typical clinical course of mustard gas lesions:

> *Conjunctivitis and superficial keratitis [inflammation of the cornea] were found in many of the patients, but the effects disappeared in about a month.*

On August 24th Hachiya describes another case of blindness in a patient who has been suffering of 'radiation sickness':

> *Mr. Sakai died, complaining of shortness of breath and blindness.*

The most likely explanation in this case seems retinal bleeding, as described and explained by Flick (see Section 8.2.3). Overall, therefore, clinical and patho-

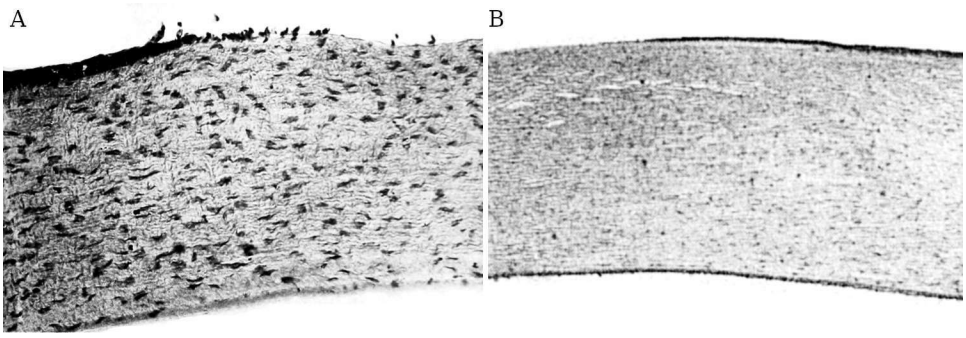


Figure 8.5 Denuded corneal epithelium. A: corneal denudation in a rabbit eye experimentally exposed to mustard gas [98]. B: corneal denudation, with regeneration underway, in a Nagasaki bombing victim [155].

logical findings on acute eye lesions don't provide any specific evidence of eye damage by ionizing radiation or by intense light, while some findings are suggestive of causation by mustard gas.

8.4 Lungs

8.4.1 Emphysema and atelectasis in early fatalities. As noted before in Section 8.1.4, the lungs have low radiosensitivity, and they should not have been significantly affected by radiation in any victims that survived the bombing for more than a day. Nevertheless, in the relatively limited number of autopsies that were performed on victims who died within the first one or two weeks, emphysema (distension of lung tissue) was commonly found: Table 8.25 in Ishikawa et al. [7] notes emphysema in 5 patients out of 12 who died between August 9th and 15th, and whose bodies were dissected by the Japanese pathologist Yamashina.

In their loot of Japanese autopsy materials, Liebow et al. [38] also observe emphysema, as well as atelectasis, which is the opposite of emphysema—namely, lung tissue that is devoid of air because it has been cut off from ventilation. They find both in the majority of the limited number of early fatalities they survey. On page 856, they note:

> *The foci of pulmonary emphysema and atelectasis without hemorrhage observed in some of the early casualties (Fig. 20) are difficult to interpret. These were found frequently at death in patients who had not been exposed to blast.*

Liebow's Figure 20 (referred to in the quote) is shown here as Figure 8.6. The deceased patient is a thirteen years old boy, who is said to have died on the

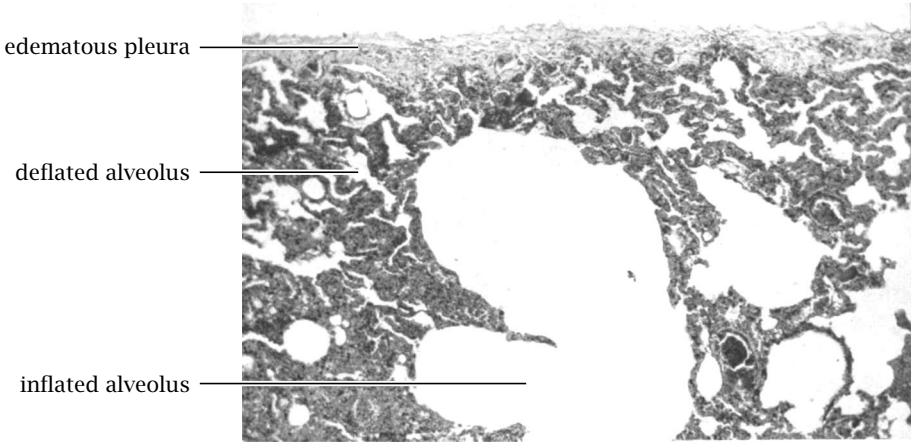


Figure 8.6 Lung emphysema (excessive inflation) and atelectasis (excessive deflation) in an early fatality from Hiroshima. The case Photograph from Liebow et al. [38], annotations by this author.

third day; thus, the lesions are truly acute and indicate some sort of obstruction of bronchioles (small bronchi).[15]

The difficulty which Liebow and colleagues perceived with interpreting their findings is readily dispelled if we consider causes other than atomic bombs. The book *The residual effects of warfare gases* [126] discusses the effects of mustard gas on the lungs and observes:

> *Emphysema was frequently found in combination with bronchitis. It usually appeared immediately after gassing and was compensatory in character, due to the extensive atelectasis found following gassing with mustard.*

The atelectasis, in turn, is understood to arise from bronchial obstruction. Thus, what we have here is a milder expression of the pathological changes in the lung that we invoked in Section 8.1.4 to account for the clinical picture in the fatalities on the day of the bombing.

8.4.2 Focal and confluent hemorrhage, inflammation, and necrosis of the lungs in later fatalities. The largest group of patients whose autopsy materials were surveyed by Liebow et al. were those who had succumbed within weeks 3 to 6 after the bombing. In slightly more than half of these cases, the

[15] If large bronchi rather than small ones had been occluded, correspondingly large segments of air-filled tissue should been cut off from ventilation, and we should not see the alternation of inflated and deflated alveoli across distances as short as evident in this picture.

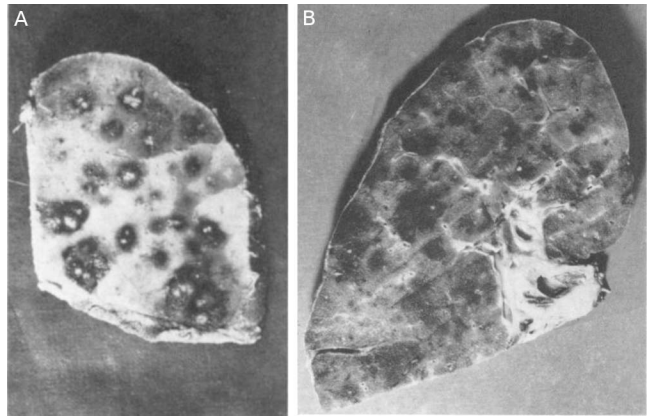


Figure 8.7 Focal necrosis, inflammation, and hemorrhage in the lungs of bombing victims. Photographs taken from Figures 17 (A) and 19 (B) in [38].

authors find a varied picture with edema, hemorrhage, necrosis, and infection. These processes were focused on the bronchioles (small bronchi) but tended to expand and become confluent (see Figure 8.7).

With respect to this group of patients, Liebow et al. don't express any puzzlement as to the causation; presumably, they ascribe their findings to the bone marrow suppression, which would pave the way for infections and also for the hemorrhage. This is indeed most likely an important contributing factor, and it would be equally well explained by radiation and mustard gas. We may note that the lesions remain centered on the bronchi, which suggests primary damage to them; this would be expected with mustard gas, yet not impossible in its absence. The same combination of findings was reported in a series of autopsies of German mustard gas victims (from the final months of World War 1) by Heitzmann [24]. In summing up his findings, Heitzmann describes the appearance of the lungs as *bunt*, that is, checkered, which seems an apt description of the lungs shown in Figure 8.7. Overall, while Liebow's findings *suggest* causation by mustard gas rather than by radiation, the time elapsed between trauma and death means that this evidence is more ambiguous than the atelectasis and emphysema at the very early stage.

8.5 Neck organs

In most of the cases surveyed by Liebow et al. [38], death occurred in or after the third week. Therefore, as with the lung pathology in the preceding section, it can be difficult to distinguish primary damage from secondary effects of bone marrow suppression, which paves the way for severe infection in these locations

and would by the third week have reached its peak. In some of their cases, however, they do describe and depict injury that is predominantly necrotic rather than infectious. This is particularly clear in the single case of early death, a young man of 19 years who died on the tenth day and who is listed in their records as 'K-98'. Concerning this patient, the authors note:

> *In the records of the necropsies of 2 individuals, K-98 (group I) and K-109 (group II), who were recently dead, the skin was said to have "peeled" easily revealing a pink raw surface beneath. The tongue, pharynx, and esophagus of one of these patients, K-98, showed remarkable changes in the epithelium with sloughing over large areas.*

Later on, they remark that "The changes in the tissues of K-98 undoubtedly represent radiation effects." Of course, these findings represent *anything but* radiation effects, since all of the epithelial tissues in question are quite radioresistant and thus should not have been destroyed more severely than any others; and it is difficult to believe that Liebow and Warren, after studying these matters for many years, would not have known this. Instead, these findings are highly suggestive of mustard gas exposure and inhalation. Liebow et al. also note that bone marrow damage has already set in, which rounds out the picture.

8.6 Other organs

Can't really think of anything right now that merits detailed discussion.

We have already covered the intestinal organs (diarrhea) briefly before. None of the other organs would be as directly and massively exposed to mustard gas as the ones discussed so far; and in quite a few organs, such as bone marrow, spleen, and gonads, the pathology is indeed very similar with mustard gas and radiation. Predictably, these are the organs that Liebow et al. [38] like to dwell on. In some others, the pathological findings are more suggestive of mustard as poisoning than of radiation, but none I can think of stands out as completely unambiguous and decisive.

A. A primer on ionizing radiation and radioactivity

This chapter is intended solely to explain some fundamental scientific concepts that will be used in later chapters; it does not focus on any specifics about the atomic bombs purportedly dropped on Japan. Readers with the required scientific background may safely skip it.

A.1 Atoms and subatomic particles

Radioactivity involves the building blocks of individual atoms, so this is where we will start our guided tour. Each atom has a nucleus, which contains one or more protons and zero or more neutrons, and it also has a shell, which contains electrons (Figure A.1). The number of protons in the nucleus determines which chemical element the atom belongs to. The atoms of a given chemical element may, however, differ by the number of neutrons; atoms of the same element that also share the same number of neutrons belong to the same *isotope*. For example, hydrogen has three isotopes, each of which has one proton. Protium, the most abundant hydrogen isotope, has no neutrons; deuterium and tritium have one and two neutrons, respectively. Nuclei that share the same number of protons and neutrons are also said to belong to the same *nuclide*.[1] This term is synonymous with 'isotope' but typically used when the focus is on the properties of atomic nuclei, rather than on specific chemical elements; for example, Figure A.1 illustrates three different nuclides.

A common shorthand notation for the composition of a nuclide uses the symbol of the chemical element, for example H for hydrogen, prefixed with a subscript that indicates the number of protons and a superscript for the number of *nucleons*, by which we mean both protons and neutrons. For example, the isotopes of Hydrogen are ${}^{1}_{1}\mathrm{H}$, ${}^{2}_{1}\mathrm{H}$, and ${}^{3}_{1}\mathrm{H}$, while the two major isotopes of uranium are ${}^{238}_{92}\mathrm{U}$ and ${}^{235}_{92}\mathrm{U}$. Since the number of protons is also implicit in the

[1] This definition of 'nuclide' ignores some finer distinctions that have to do with different energetic states of atomic nuclei. There will be many more instances of simplified treatment in this chapter, which is intended for quick orientation only but not as a definitive reference.

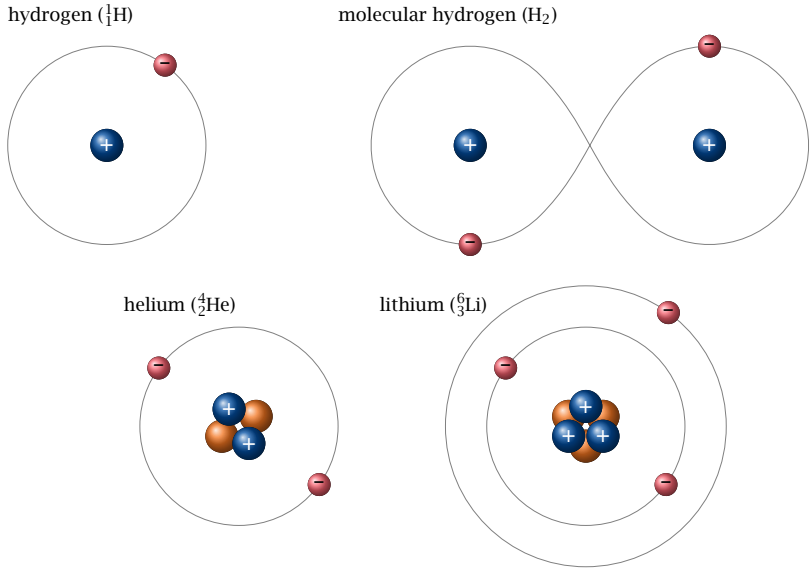


Figure A.1 Bohr model of atomic structure. The atom consists of protons (blue), neutrons (orange), and electrons (red). Protons and neutrons are located in the nucleus; they have similar mass, but only the protons carry a positive charge. Prefixed subscripts indicate the number of protons, and superscripts the sum of protons and neutrons (i.e., nucleons). Electrons are negatively charged and are found in the shell. They prefer to form pairs, either within single atoms (e.g. helium, He) or within molecules composed of two or more atoms (e.g. H_2). See text for further details.

element, the corresponding prefix is often omitted, as in ^{235}U instead of $^{235}_{92}$U or ^{3}H instead of $^{3}_{1}$H.

Protons and neutrons are very similar in mass but differ in electric (coulombic) charge. Neutrons are uncharged, whereas each proton carries a single positive charge. The magnitude of this charge equals that of the electron; however, the latter's charge is negative. In the common case that the number of protons in the nucleus equals that of the electrons in the shell, the atom has no net charge. On the other hand, if the atom is short of electrons or has surplus ones, it will have a positive or negative net charge. Atoms (and also molecules) that are in a charged state are called *ions*.

A.2 Chemical bonds and molecules

In everyday chemistry—including biochemistry, that is, the kind of chemical reactions that occur in the human body and other living organisms—only the electron shells of the atoms take an active part; the nuclei are merely passengers. There is a number of rules that govern the behavior of the electrons, and

therefore the chemical reactivity of each element. One of these rules states that electrons prefer to form pairs. If all electrons of an atom can form pairs within that atom's shell, then the element in question typically has low reactivity. An example is helium (shown in Figure A.1), which occurs in nature as a one-atomic gas. On the other hand, hydrogen and lithium have unpaired electrons in their shells, and they are therefore more reactive. Two hydrogen atoms can mutually satisfy their preference for electron pairing by sharing their electrons orbit within in a joint, dumbbell-shaped orbit (the chemical term is *orbital*). The shared electron pair constitutes a chemical bond between the two hydrogen atoms, which thus have become a single hydrogen molecule (H_2). Lithium can react analogously with other atoms, although two lithium atoms will not form a stable molecule.

The atoms of some elements have more than one unpaired electrons in their shells; for example, oxygen has two, and nitrogen has three. With nitrogen, all of these these can be paired in a diatomic nitrogen molecule (N_2). To indicate that this molecule contains three shared electron pairs or bonds, N_2 may be written as $N \equiv N$, while H_2 with its single bond is represented by H – H.

In contrast to nitrogen, molecular oxygen (O_2) does not manage to properly pair all electrons; its electronic structure may be written as $^{\bullet}O - O^{\bullet}$ to indicate that one stable electron pair is formed, while the other two electrons, represented by the dots, remain 'lonely.' This difference in internal electron pairing explains the very different reactivities of oxygen and nitrogen, for example *vis-a-vis* hydrogen: while N_2 can be coaxed into reacting with hydrogen only at very high pressure and temperature,[2] oxygen requires only a spark to explosively react with hydrogen. The product of the reaction ($2\,H_2 + O_2 \longrightarrow 2\,H_2O$) is of course water; its bond structure may be written as H – O – H, which means that in this molecule all the electron pairing needs of oxygen are satisfied. Water is therefore a fairly stable molecule. Oxygen also reacts with carbon (C) to form a stable product, carbon dioxide (CO_2, or O = C = O), again with the release of energy; and similarly with many other elements. The wide scope of oxygen's reactivity is reflected in the familiar observations of combustion and corrosion.

The association between unpaired electrons and chemical reactivity is not limited to the oxygen molecule. Below, we will see that ionizing radiation can break up electron pairs within initially stable atoms and molecules, which thereby become reactive. Before considering the biological significance of this effect, we will first consider the physical basis of radiation and radioactivity.

[2] The reaction of molecular nitrogen and hydrogen at high pressure and temperature—$N_2 + 3\,H_2 \longrightarrow 2\,NH_3$, with NH_3 representing ammonia—is the Haber-Bosch process. It is industrially important for the production of nitrogen-based fertilizers and explosives.

A.3 Radioactivity

While chemical reactivity is determined by the electron shell, radioactivity is a property of the atomic nucleus alone. Most of the atomic nuclei that occur in nature are stable, but some are not; these will at some point in time decay. The stability of a nucleus depends on the ratio of neutrons to protons contained in it, as well as on its overall size, that is, its overall count of protons plus neutrons.

We have already encountered the three isotopes of hydrogen (see Section A.1). Protium and deuterium are stable, whereas tritium is not, because it has too many neutrons. It therefore decays through the emission of an electron (e^-):

$$^3_1\mathrm{H} \longrightarrow {}^3_2\mathrm{He} + e^- \tag{A.1}$$

The emission of the negatively charged electron is balanced by changing one neutron to a proton, which creates a positive charge. The neutron excess is thereby remedied; the resulting nucleus, which now belongs to a different element (Helium, He), is therefore stable.[3]

The electron produced by the decay is catapulted out of the nucleus with considerable energy, which it will dissipate by colliding with atoms and molecules in its path. The energy transferred in these collisions causes additional electrons to be ejected from those atoms and molecules. This will turn the target atoms and molecules into ions, and with molecules it may also break chemical bonds. The formation of ions along the emitted particle's path can be readily detected; hence, this phenomenon is known as *ionizing radiation*, and nuclides that produce it are known as *radioactive*.

A.3.1 Radioactive half-life and activity. The exact time at which an individual nucleus will decay is unpredictable, but the probability that it will decay within a certain time period can be determined, and this is a fixed and characteristic property of the isotope in question. Processes that follow this pattern—decay or conversion of a species at a rate that is directly proportional to its own abundance—can be described by an exponential function:

$$N_t = N_0\, e^{-t/\tau} \tag{A.2}$$

In this equation, N_0 is the number of atoms at time zero ($t = 0$), and N_t is the number remaining after some time interval t. The *time constant* τ is the time required to reduce a given number of atoms (N_0) of the nuclide in question to

[3] The reaction also releases an antineutrino (written as $\overline{\nu_e}$), a subatomic particle with no charge and very small mass. It will carry off a substantial share of the energy released in the decay, but it is otherwise inconsequential in the context of biological radiation effects.

the residue N_0/e. Alternatively, we can use the nuclide's *radioactive half-life* ($t_{1/2}$), which is the time required to reduce N_0 by half.[4] In the case of tritium, the half-life is 12.3 years.

Equation A.2 states that the residual number N_t of a nuclide is an exponential function of time. The first derivative of N_t is the *activity* (A_t) of the nuclide:

$$A_t = \frac{\mathrm{d}N}{\mathrm{d}t} = -\frac{N_0}{\tau}\, e^{-t/\tau} \tag{A.3}$$

The activity is measured in units of seconds^{-1}, which in this context[5] is referred to as *Becquerel* (Bq):

$$1\mathrm{Bq} = 1\mathrm{sec}^{-1}$$

The relationship stated in (A.3) is illustrated in Figure A.2 for three hypothetical nuclides, which at $t = 0$ are present at the same amounts (N_0). Because the time constant occurs in the denominator of the pre-exponential term, the nuclide with the smallest time constant—or the shortest half-life; in our example, one day—shows the highest activity per quantity of nuclide, or *specific activity*. However, after 20 days—that is, 20 successive half-lives—its activity has dropped to about one millionth of the initial value. On the other hand, the nuclide with the longest half-life (100 days) is still present at appreciable levels even after 200 days.

The half-lives of nuclides occurring in nature or in artificial nuclear reactions vary to a much greater extent than those in our example—namely, from fractions of a second to billions of years. According, they have vastly different specific activities. Some of the nuclides that are formed when a nuclear bomb explodes have very short half-lives, and thus cause a 'flash in the pan' with very high activity for a very short time, sometimes lasting no longer than the blast itself. Others can be detected for many years afterwards, but because of their relatively low specific activity don't contribute significantly to the acute radiation dosage.

A.3.2 Types of radioactive decay. The form of decay observed with tritium—conversion of a neutron to a proton, with the ejection of an electron from the nucleus—is very common, and it is particularly important with the fission products of uranium and plutonium (see later). It is referred to as β-decay, and more specifically as β^--decay, since the ejected electron is negatively charged.

[4] There is a simple relationship between both time parameters: $t_{1/2} = \ln 2\, \tau \approx 0.693\, \tau$. Furthermore, the inverse of τ is defined as the rate constant, k. Thus, we can write $N_t = N_0\, e^{-k\tau}$.

[5] In the context of wave frequencies, the same basic unit (second^{-1}) is named Hertz (Hz).

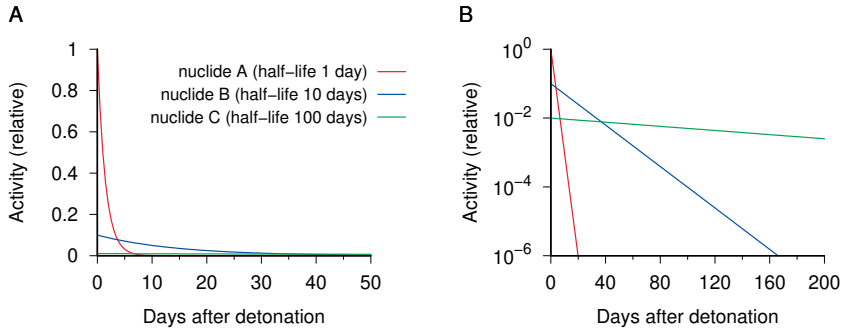


Figure A.2 Time course of activity for three hypothetical nuclides with different half-lives. At time zero, the amounts of nuclides A-C are identical, but the activity is highest for nuclide A, which has the shortest half-life. After three weeks, however, A is practically gone, and after 200 days only nuclide C is still present at appreciable levels. Panels A and B depict the same hypothetical decays, but the semilogarithmic plot format in B better displays activities with very different magnitude.

Some nuclides that undergo β-decay may concomitantly also emit a neutron. While this is comparatively rare, it does occur among the fragment nuclei that result from nuclear fission, and these neutrons form part of the neutron radiation released by nuclear bombs.

In many cases, a nucleus undergoing β-decay does not get rid of all available energy in the process. In these cases, the remainder is emitted, usually a short time later, as a γ-particle, which is a *photon*—a particle of the same nature as light, but with much higher energy (and correspondingly shorter wavelength). γ-Particles, or γ-rays, can also be produced by nuclei that need to offload surplus energy originating from other processes, including α-decay, nuclear fission, or the non-elastic collision with neutrons (see below).

While the nuclei of tritium and of most nuclear fission products contain too many protons for stability and thus undergo β^--decay, the opposite case also occurs. Unstable isotopes that have too few neutrons may achieve stability by 'reverse' β-decay, or *electron capture*. Here, the nucleus picks up an electron from the atomic shell, and one of the protons is thereby converted to a neutron. The nucleus may again release excess energy through γ-radiation. An example is the iodine isotope ^{125}I, which decays to an isotope of tellurium (Te):[6]

[6]The γ-radiation emitted by ^{125}I is very convenient to work with. It is soft enough to be easily shielded with a little bit of lead, yet hard enough not to be trapped inside inhomogeneous samples, and the lifetime of the isotope (59 days) offers a good trade-off between sensitivity and sample stability. Moreover, it is easy to couple ^{125}I to protein or drug molecules of interest. It is therefore widely used as a tracer in biochemical experiments.

$$^{125}_{53}\mathrm{I} + e^- \longrightarrow {}^{125}_{52}\mathrm{Te} \tag{A.4}$$

In α-decay, the emitted particle is larger and heavier than in β-decay—it contains 2 protons and 2 neutrons, and therefore is identical with the nucleus of the stable helium isotope ^{4}He. α-Decay is particularly important with very heavy elements[7] such as radium, thorium, uranium, and the artificially produced elements that exceed the atomic number—that is, the proton count—of uranium. These 'transuranes' include in particular plutonium, which is formed from the uranium isotope ^{238}U through neutron capture and two subsequent β-decays (see below). α-Decay may also be accompanied by the release of γ-radiation.

A.3.3 Decay chains. The products of radioactive decay may themselves be unstable and decay in their turn, and successive decays may form a chain that continues for multiple generations. An important natural decay chain begins with $^{238}_{92}$U and ends with lead ($^{206}_{82}$Pb), which is stable. The total number of nucleons declines by 32, which corresponds to 8 α-particles overall. 8 α-Decays would reduce the number of protons by 16, but the actual difference is only 10, which means that 6 protons must be converted to neutrons through β-decay. Accordingly, the total number of α- and β-decays is 14.

The half-life of ^{238}U, at 4.47 billion years, is much longer than those of all intermediate species. This has the interesting consequence that the *activities*, that is, the number of decays per unit of time, of all chain members in a natural, undisturbed uranium ore sample will be virtually equal. To see why, assume that you start with a sample of pure ^{238}U. As the uranium undergoes α-decay with very low, virtually constant activity, its daughter nuclide (^{234}Th, an isotope of thorium) will accumulate. ^{234}Th has a half-life of only 24 days and will therefore decay rapidly; it can accumulate only until its own decay rate reaches the rate of its formation, which is of course identical with the activity of ^{238}U. The same principle applies to all of the other decay intermediates, including the uranium isotope ^{234}U, which is formed two β-decays downstream of ^{234}Th. Therefore, in natural uranium, the activities of ^{238}U and ^{234}U should be equal, even though ^{234}U is much less abundant.[8] We will make use of this relationship in Chapter 2.

[7] The word 'heavy' in this context refers to the mass of individual nuclei rather than the density of the element as a solid material. However, both are correlated—elements with heavy nuclei also have high densities.

[8] Isotopes of the same element tend to differ slightly in solubility etc., which may cause them to be differentially enriched or depleted for example in soil samples or in biological samples, which will then cause their activities to differ slightly—they are approximately equal, but not exactly.

A.4 Interaction of ionizing radiation with matter

As briefly stated above, all types of particles released by radioactive decay will cause ionization: as they collide with atoms and molecules along their path, they will transfer some of their initially ample energy to the electrons of those targets, and the electrons will thereby be ejected from their atomic shells, turning the atoms and molecules into ions. Since these ions are readily observed in *ionization chambers* (see below), all of these disparate particles came to be known as 'ionizing radiation'. However, these particles cause other effects beyond ionization, and some of these affect living organisms.

A.4.1 Radical formation. Ejection of electrons can happen not only with individual atoms but also with molecules, which may thereby be broken up. A straightforward example is the cleavage of water molecules, which may be written as

$$\mathrm{H{-}O{-}H} \longrightarrow \mathrm{H^+} + e^- + {}^\bullet\mathrm{O{-}H} \tag{A.5}$$

What happened here? One electron (e^-) that was part of an O – H bond has been ejected. The hydrogen atom has been ionized (H^+), and the second bond electron is retained by the residue of the molecule ($^\bullet$O – H, or $^\bullet$OH), whose dot represents this now unpaired electron. An atom or molecule with an unpaired electron is referred to as a *radical*.

Due to their unpaired electrons, radicals tend to be highly reactive, and none is more so than the hydroxyl radical ($^\bullet$OH). Since water is abundant in living organisms, $^\bullet$OH is a prominent product of irradiation and the most important mediator of its deleterious effects (see later).[9]

A.4.2 Interactions of γ-rays with matter. For the most part, γ-rays cause ionization and radical formation as described above. Most commonly, the interaction with electrons in target atoms will take the form of *Compton scattering*—the γ-photon collides with an atom or molecule and ejects one of its electrons. In the process, it also transfers some of its kinetic energy to the atom, which causes the γ-photon itself to change direction. This can repeat a number of times until the energy of the γ-photon is depleted.

Since γ-rays dissipate their energy through successive collisions with electrons, it follows that sufficiently thick layers of matter, which contain a large enough number of electrons, can act as a shield against γ-radiation. Since

[9] The oxygen molecule ($^\bullet$O – O$^\bullet$) is a radical, too, and it can react like one. For example, if you have ever patched a bicycle tire, you have observed radical polymerization induced by molecular oxygen.

atoms contain equal numbers of electrons and protons, heavy elements make particularly good shields; lead is very commonly used for this purpose.[10]

A.4.3 Interaction of α- and β-particles with matter. Due to their slower speed and their electric charge, α- and β-particles interact with electrons more effectively than do γ-rays. Thus, after entering a target, both particle types produce many secondary ions in rapid succession, at a high local density, and in doing so quickly exhaust their energy. They therefore do not penetrate matter very deeply (see Section A.7.1).

A.4.4 Neutron interactions with matter. Unlike the other particles considered here, neutrons don't interact with electrons directly, but only with atomic nuclei. The collision of a neutron with a nucleus may have three different outcomes:

1. the neutron may bounce off, such that the overall amount of kinetic energy is preserved, but some part of it is transferred to the nucleus. This is known as *elastic neutron scattering*;
2. it may be 'swallowed up' by the nucleus. This is known as *neutron capture*;
3. it may be captured briefly but immediately ejected again. This is referred to as *non-elastic neutron scattering*.

When neutrons of sufficient energy are scattered elastically by hydrogen nuclei, the latter will be yanked loose from the molecules that they are part of and sent flying; these so-called 'recoil protons' then cause the actual ionization and radical formation. This effect mediates most of the biological effects of neutron radiation and also is important for its detection.

Virtually any nuclide can capture a neutron, but the probability varies both with the composition of the target nucleus and the kinetic energy of the neutron. With most nuclides, neutrons of low energy—called *thermal neutrons*, since their kinetic energy is in equilibrium with the surrounding atoms, whose kinetic energy reflects the temperature of the system—are captured the most readily. Figure A.3 illustrates how the probability of capture varies with the energy of the neutron on the loose with two different nuclides, cobalt-59 and uranium-235.

[10] Another interesting effect that occurs with γ-photons of sufficiently high energy is that of *pair production*—the γ-photon is converted to an electron-positron pair ($e^- + e^+$). The positron will swiftly bump into another electron, which will cause annihilation of both particles and give rise to two γ-photons. Thus, for practical purposes, pair-production can be considered a transitory stage in the dissipation of γ-ray energy.

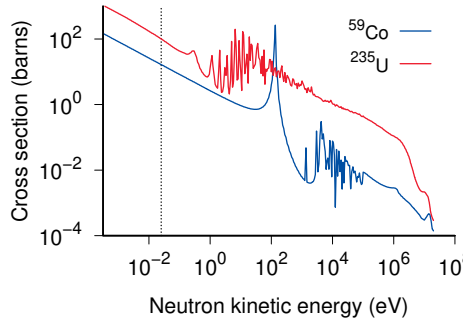


Figure A.3 Neutron capture cross sections of ^{60}Co and ^{235}U, as a function of neutron kinetic energy. The cross section has the dimension of an area but really measures the probability of capture. The black dotted line indicates the typical energy of a thermal neutron (0.025 eV). Data taken from [157].

These two neutron capture reactions can be written as follows:

$$^{59}_{27}\mathrm{Co} + n \longrightarrow {}^{60}_{27}\mathrm{Co} \tag{A.6}$$

$$^{235}_{92}\mathrm{U} + n \longrightarrow {}^{236}_{92}\mathrm{U} \tag{A.7}$$

The products of neutron capture are often unstable, and this is the case with both of the above examples. ^{60}Co undergoes radioactive β- and ɣ-decay with a half-life of 5.27 years. The ɣ-particles emitted by ^{60}Co are quite high in energy; they can be used e.g. for the irradiation treatment of cancer or for sterilizing medical equipment. With ^{236}U, most nuclei immediately undergo fission (see below); however, a minor fraction of nuclei don't fission but instead 'simmer down' and undergo radioactive decay with a very long half-life (23.4 million years).

In both the capture and the non-elastic scattering of neutrons, the atomic nuclei are transiently promoted to more energy-rich states; they release this surplus energy in the form of ɣ-radiation. These secondary ɣ-rays contributes to the biological effects of neutron radiation.

A.5 Nuclear fission

As an alternative to α- or β-decay, some unstable nuclides may undergo nuclear fission. In this process, the nucleus breaks up into two large fragments of somewhat variable size and composition, plus two or three individual neutrons. Most of the nuclear energy released by the fission is converted to kinetic energy, causing the two fission fragments and the neutrons to dash off like scalded cats; some more energy is released in the form of ɣ-radiation.

Some nuclides may fission spontaneously, while others fission only upon neutron capture. Among the latter, some are fissioned only by neutrons of high energy, whereas others are readily fissioned by any neutrons at all, regardless of their kinetic energy. This leads to the following distinction:

1. a *fissionable* nuclide releases neutrons which are too low in energy to fission another nucleus of the same nuclide.
2. a *fissile* nuclide releases neutrons which *can* fission another nucleus of the same nuclide; thus, with these nuclides, fission can potentially occur as a chain reaction.

Among the isotopes of uranium, ^{238}U is fissionable, whereas ^{235}U is fissile. ^{235}U is indeed the *only* fissile nuclide with useful natural abundance; however, additional ones can be produced artificially from certain precursor nuclides; these are called *fertile*. The most important fertile nuclides are ^{238}U and ^{232}Th, which upon neutron capture undergo two sequential β-decays to turn into the fissile nuclides ^{239}Pu and ^{233}U, respectively.[11] While ^{232}Th is more abundant than ^{238}U, there are some technical obstacles to the use of its fissile derivative ^{233}U as bomb material. This leaves ^{235}U and ^{239}Pu as candidates for such use; the Hiroshima bomb ('Little Boy') is said to have contained ^{235}U, whereas the Nagasaki bomb ('Fat Man') purportedly contained ^{239}Pu.

A.5.1 Products of nuclear fission. Each fissile nuclide gives rise to a *distribution* of fission products rather than two distinct species. The shape of the distribution varies somewhat between nuclides and also with the energy of the neutrons that bring about the fission; in particular, it differs between nuclear reactors and bombs, which use low and high energy neutrons, respectively. Figure A.4 shows the distributions produced by ^{235}U and ^{239}Pu when fissioned with fast neutrons, that is, under conditions similar to those that would prevail in a fission bomb. The fission products fall into two clusters centered at approximately 140 and 95 nucleons, respectively. Both nuclides produce a very similar amount of ^{137}Cs, which was already introduced in Chapter 1 as a marker of fallout in environmental samples. In both cases, ^{137}Cs is produced in approximately 6% of all fission events; thus, from the abundance of ^{137}Cs in the fallout, it is possible to estimate the total amount of bomb fuel that must have fissioned.

[11] You may notice that ^{238}U is both fertile and fissionable. The outcome depends on the energy of the captured neutron; fast neutrons will induce fission, while slow ones will initiate conversion to ^{239}Pu.

In so-called 'breeder' reactors, fissile and fertile nuclides are mixed on purpose, and a fraction of the neutrons produced by the ongoing chain reaction is used to 'breed' more fissile nuclides for use as reactor fuel or bomb material.

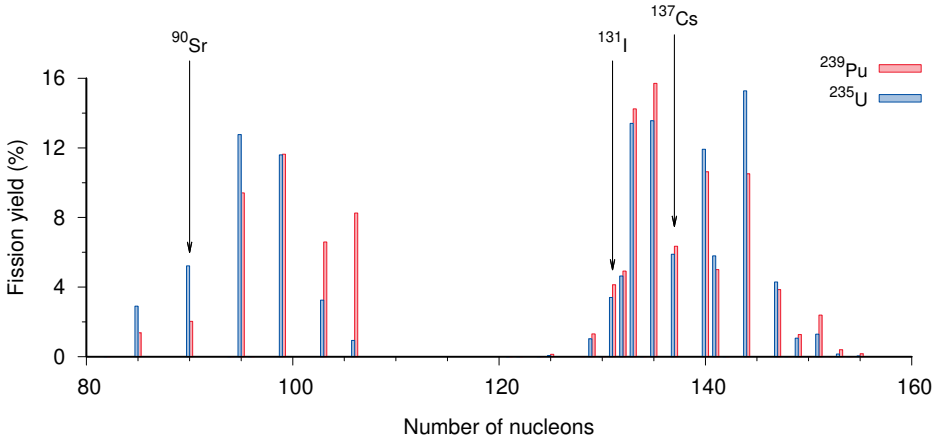


Figure A.4 Fission products of ^{239}Pu and ^{235}U when fissioned by fast neutrons. Nuclides with the same numbers of nucleons were lumped together in this graph, but the three nuclides highlighted in this graph all have unique nucleon numbers. ^{90}Sr chemically resembles calcium and accumulates in bone, whereas ^{131}I accumulates in the thyroid gland. ^{137}Cs resembles potassium and may accumulate diffusely in tissues. In addition, it is also commonly used as an environmental marker of nuclear fallout. Data from https://www-nds.iaea.org/sgnucdat/c3.htm.

^{131}I (iodine) and ^{90}Sr (strontium) are fission products that may accumulate in specific organs and potentially cause disease. ^{90}Sr chemically resembles calcium and accumulates in bone mineral; its proximity to the bone marrow may contribute to the causation of leukemia. Its half-life is 28.8 years, which means that it remains detectable in the bone for significant lengths of time. In contrast, the half-life of ^{131}I is only about a week. This is nevertheless long enough for it to be dispersed with the fallout and to accumulate in thyroid gland tissue. ^{131}I released in the Chernobyl disaster caused numerous cases of thyroid cancers in the adjacent areas of Ukraine and Belarus [158].

Another point to note is that fission products such as the three discussed above will typically form directly. Instead, the immediate fission products tend to be very short-lived and decay into longer-lived ones through one or more β-decays; this is illustrated in Figure A.5. The γ-rays emitted as part of these secondary decays contribute significantly to the immediate radiation of the bomb. Some of these decay events will also release neutrons; while these make only a minor contribution to the bomb radiation, they are crucial for controlling chain reactions inside nuclear reactors.

A.5.2 Fission bombs. The detonation of a fission bomb occurs through a chain reaction, which starts when the first ^{235}U or ^{239}Pu atom captures a neutron—

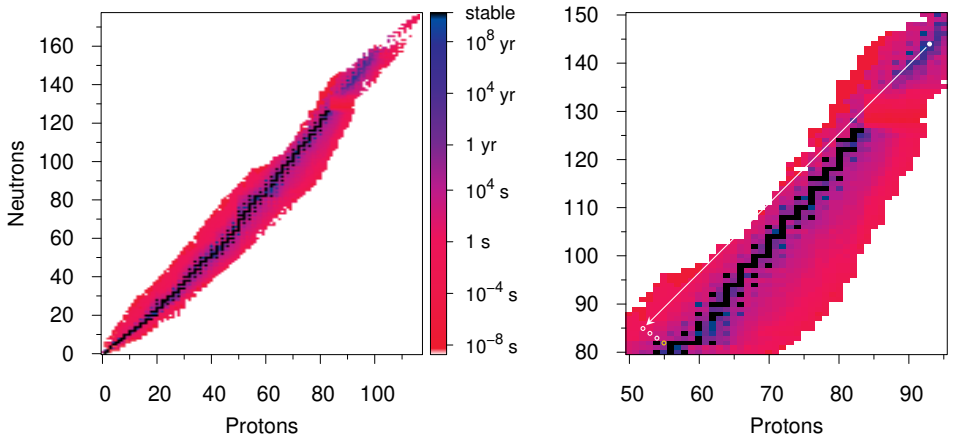


Figure A.5 Nuclear stability as a function of proton and neutron numbers. Left: For almost all proton numbers (or elements) up to 82, there is at least one neutron number that will result in a stable nucleus (black). Radioactive isotopes with long half-lives (blue shades) are typically found close to this relation of stability, which is curved slightly upwards. Right: among the multitude of ^{235}U fission products, there may be a nuclide with 52 protons and 85 neutrons (arrow). Within seconds, this highly unstable species will undergo three successive β-decays to become ^{137}Cs (yellow circle). While still radioactive, ^{137}Cs is long-lived enough to be found in the fallout.

supplied by a small neutron source built into the bomb—and undergoes fission. This produces two fragment nuclei and 2 or 3 neutrons. Each of the neutrons can potentially be captured by another fissile nucleus and cause it to fission in turn. The likelihood of such secondary fission events depends on the number of fissile nuclei within reach of each liberated neutron. Once this likelihood becomes so high that, on average, each fissioning nucleus will give rise to more than one fission event in the next generation, the chain reaction will be rapidly amplified and cause the detonation.

From the foregoing, we can understand in outline what the consequences of a nuclear detonation will be. The copious kinetic energy of the fission products and neutrons is converted to heat. The heat produces a flash of light, and it also drives expansion of the surrounding air, which gives rise to a pressure shock wave. Much of the γ-rays and some of the neutrons will escape from the detonating bomb core and cause an intense pulse of ionizing radiation. In contrast, the β-particles released by short-lived fission intermediates have only short free path lengths and remain confined within the core. In summary, the immediate long-range effects of a detonating fission bomb comprise intense radiant heat, a shock wave, and ionizing γ- and neutron radiation.

A.6 Ionizing radiation unrelated to radioactivity

The particles released by radioactive decay are ionizing primarily due to their high energies; the source of that energy—in this case, the atomic nuclei undergoing decay—is not important. Other, artificial means for endowing particles with similarly high energies exist, and the energy-rich particles thus generated will be every bit as ionizing as those arising from radioactivity.

There is no need for a comprehensive survey in the context of our subject, but some examples are relevant and useful. The process always begins by accelerating a charged particle in a vacuum using high voltage. The easiest such process involves the acceleration of electrons, which then strike a metal target. Within that target, they will collide with other electrons, to which they will transfer some of their energy, which is then released in the form of X-rays, which are electromagnetic radiation of high energy. The photon energy of this radiation is determined by the strength of the electric field used for electron acceleration, and it can match or even exceed the photon energy of γ-rays. Such high-energy X-rays can be used interchangeably with γ-rays in technical or medical applications. Similarly, the accelerated electrons themselves can be used to mimic β-radiation.

The artificial generation of neutrons in the laboratory can be accomplished by stripping some atomic nucleus of electrons and using an electric field to accelerate it and slam it into another nucleus. Most commonly, this is done with two isotopes of hydrogen (deuterium and tritium); the collision of the two nuclei will produce helium and a free neutron. In the early days, including those following the alleged atomic bombings, the production of neutrons in quantity required cyclotrons, but in the meantime smaller, simpler devices have been invented. Such artificial neutron sources can be used to mimic, and thus to study, the effects neutron radiation from atomic bombs.

The process of charged particle acceleration by an electric field also makes plain the meaning of the physical unit electron volt (eV)—it is equivalent to the energy which an electron, or another particle with a single charge, will acquire when traveling through a vacuum from one electrode to another when a potential of 1 V exists between the two. The energies of particles released by radioactive decay are typically stated in kilo-eV (keV) and mega-eV (MeV). For example, the decay of ^{60}Co produces β-radiation with 317 keV as well as γ-radiation with 1.17 MeV and 1.33 MeV. We can mimic those β-particles by sending electrons down a field with 317 kV, and the γ-radiation by accelerating electrons using

1.17 or 1.33 MV and then converting them to X-rays by slamming them into a metal target.[12]

A.7 Attenuation of ionizing radiation by matter

When a particle of ionizing radiation impinges on some target matter, it will begin to ionize the atoms and molecules within; and since each ionization event requires some energy, the ionizing particle itself will eventually run out of energy and come to rest or vanish. To what depth the particle can penetrate before this occurs obviously depends on the initial energy of the ionizing particle; in addition, however, it also depends on its nature, which determines at what range it can interact with individual electrons or nuclei in the target matter.

A.7.1 Distinctions between particle types. The interaction with the longest range is the Coulomb force; accordingly, α- and β-particles, which are electrically charged, interact the most readily and produce the greatest number of ions along a certain path length. This also means that they shed their energy very quickly and thus penetrate the target matter only to a very shallow depth. Among the two, the α-particles are heavier and slower; they thus spend more time in the vicinity of a given single electron and stand a greater chance of exerting enough pull to pry it loose from its host atom. Therefore, α-particles exhibit the highest density of ionization, which implies the shallowest depth of penetration; in fact, they cannot even penetrate intact human skin deep enough to reach the basal layer of vital, regenerating cells. Isotopes that emit α-radiation thus can harm humans only when ingested or inhaled.

The lighter and faster β-particles move faster and do not ionize quite as many atoms or molecules along a given stretch of path within the target, which results in somewhat deeper penetration. Even they, however, will only penetrate human skin to a depth of a few millimeters; thus, while while β-emitting radionuclides may burn the skin from without, they may cause damage to interior organs only after they have been taken up. This is illustrated by the aforementioned fission products ^{131}I and ^{90}Sr, which will cause disease only after accumulation in the thyroid gland or bone matrix, respectively.[13]

In contrast to α- and β-particles, γ-photons have no charge, and they thus will interact with electrons only when they hit them straight on. Thus, on average, a γ-photon will travel a much longer distance between two consecutive

[12] Note that in this case some, but not all the X-ray photons will receive the full amount of energy. A better way to mimic energetically homogeneous γ-rays is through synchrotron radiation.

[13] It is, however, possible to achieve deeper electron penetration by accelerating them to very high energies. Such artificial high-energy electron radiation is used in the radiation therapy of cancer.

ionization events; it will shed its energy more slowly and penetrate the target to a much greater depth, or even traverse it. The depth of penetration will be inversely proportional to the number of electrons per volume segment of target matter; thus, matter that consists of comparably light atoms, for example water or soft tissues, will be penetrated most readily, whereas matter that contains heavier atoms such steel or bone mineral stop γ-rays more readily.[14]

Neutrons are uncharged as well; unlike γ-rays, they interact primarily with the nuclei of the target matter, and moreover they lose energy more readily by colliding with lighter nuclei than with heavier ones. Like γ-rays, however, they can penetrate the walls of buildings and human tissues to considerable depths. Both neutrons and γ-rays thus contribute to the total radiation dose due to a nuclear detonation.

A.7.2 Quantitative aspects of attenuation. As a first approximation, we can consider a block of matter as composed of many stacked layers of uniform thickness, and then postulate that each layer attenuates the impinging radiation by a constant fraction or percentage. This results in an exponential relationship: just as we can determine half-lives for the effect of time on radiation intensity, we can determine half-thicknesses for the shielding effect of matter. From the foregoing, it will be clear that the actual values of such half-thickness will vary not only with the type of the shielding material but also with the type and energy of the radiation. Quite commonly, one will find values tabulated for layer thicknesses that attenuate radiation by 90% rather than 50%; this latter value will be approximately 3 times greater.[15]

There are some effects that limit the accuracy of this simple approach:

- Particle energies are usually inhomogeneous, and particles with higher energies will penetrate more deeply.
- Even if the radiation is produced by a point source, its particles may be scattered rather than fully stopped; they will thus change direction and lose some of their energy.
- Some primary particles will produce secondary radiation: stopped β-particles may produce X-rays, and stopped neutrons will produce γ-rays. These secondary rays will typically be more penetrating than the primary particles that produced them.

[14] Remember that γ-rays are of the same nature as X-rays. Bones show up white on an X-ray film because the heavier elements (calcium and phosphorus) in bone mineral stop the X-rays. In contrast, the X-rays traverse the surrounding soft tissues and blacken the film.

[15] Consider that $0.5^3 = 0.125$, or 12.5%; therefore is, three stacked half-thickness layers will attenuate the radiation by 100% − 12.5% = 87.5%.

Effects like these need to be taken into account in order to accurately determine the dosages received for example by persons located inside a house, as discussed by Auxier [33]. Nevertheless, the exponential approximation is useful in practice at least for orientation. It should also be noted that it can be applied not only to solids or liquids, but also to gases, including the atmosphere; the difference is simply that the shielding half-widths in the atmosphere will be far larger.

A.8 Measurement of ionizing radiation

In order to detect and quantify ionizing radiation, we must observe some of its interactions with matter; and to do so sensitively, we must find ways to amplify the initial signal generated in this interaction. Several different physical principles are exploited for these measurements.

A.8.1 Ionization. This is observed in an *ionization chamber*, an enclosure that is filled with some noble gas and also hosts two electrodes, between which a high voltage is applied. When an ionizing particle traverses this chamber, it will collide with gas atoms and knock electrons out of their shells. In the strong electric field, the ions and the electrons will become separated and be attracted toward the two opposite electrodes, where they will cause an electric signal. The magnitude of this signal will be proportional to the number of ions that were generated; and this number will vary depending on the type and energy of the ionizing particle as outlined above.

In what form exactly the signal is received depends on the experimental setup. If the voltage between the electrodes is applied only initially but not renewed, then each detected burst of ions and electrons will decrease that initial voltage. This means that the measurement will be cumulative—we will be able to estimate how many ions were generated, but not by how many ionizing particles. If the voltage is kept constant, then the signal is the current required to restore the voltage to its preset level after each ionization event; and since this restoration will occur quickly, it will be possible to count the number of ionizing particles over a certain time interval.

Even though it may be counterintuitive, the signal can be amplified by reducing the gas pressure inside the chamber. A low pressure will reduce the number of collisions between the ionizing particle and the gas atoms, and therefore the number of ions and electrons released; however, while traveling toward their respective electrodes, these ions will gather more speed before colliding with other gas atoms, and due to this greater speed they will be able to ionize those gas molecules in turn. The overall result will be a cascading proliferation of

charged particles and therefore amplification the electric signal. There are in principle two ways to exploit this mode of amplification:

- The amplification may limited in extent, such that the final signal is still constrained by the number of ions and electrons generated directly by the ionizing particle. Then, the signal will retain information about the nature and energy of the ionizing particle.
- The amplification may be saturating—each event is amplified to the same, maximal extent, regardless of the strength of the original ionization. This will maximize sensitivity, but on the other the ability to discriminate between different particle types is lost. This latter principle is applied in the widely used Geiger counters.

Both modes of detection have their uses. Generally speaking, counting devices optimized for sensitivity tend to be simpler and are more suitable for field use. Instruments that can discriminate different particle types are more complex and mostly used in the lab. The key advantage is that particle energies can be used to discriminate and identify different radionuclides in complex mixtures such as soil, which may contain both natural background and nuclear fallout.

A.8.2 Scintillation. Like ionization, this physical effect begins with a collision between an ionizing particle with an electron of some other atom or molecule. However, in scintillation, the electron is not knocked free but only transiently promoted to a higher state of energy within its host particle. When it falls back to its initial level, the surplus energy which it received in the collision is released as light (a single photon). The light can be focused onto a photomultiplier and quantified; the intensity of the flash of light will be proportional to the number of scintillating atoms or molecules and thus to the energy of the ionizing particles. ɣ-Rays induce intense scintillation in materials such as crystalline sodium iodide, and this is exploited for their detection.

A.8.3 Thermoluminescence. Some materials, particularly ceramic ones, may show a peculiar response to ionizing radiation: the dislocated electrons may migrate through the material for some distance and become trapped in a *metastable state*, that is, a state that is high in energy, yet unlike most other high-energy states does not spontaneously fall back to a lower energy level. It can, however, be induced to give back its energy in the form of light by heating the material. This heat-induced light signal is called *thermoluminescence*.[16]

[16]For a simple analogy, consider a pinball machine. The plunger is the ionizing particle, and the ball is the electron. When you pull the plunger, the ball receives energy and starts rolling. Most of

The metastable state can persist for potentially very long periods of time, which means that it gives the material a 'memory' for the ionizing radiation it was exposed to in the past. Ceramic material is fairly dense and thus will not be significantly penetrated by α- or β-radiation. Neutrons and γ-rays may penetrate it, but of these only γ-rays interact with electrons effectively; thus, in practice, all the observed thermoluminescence activity can be ascribed to γ-rays.

An interesting application of thermoluminescence concerns the dating of ceramics recovered in archaeological excavations [159]. Firing a new piece of pottery will purge the clay of any previously accumulated luminescence energy and thus 'reset the clock', and repeated heating on a fire while in use will do the same. Once it becomes emplaced underground, however, its pent-up thermoluminescence will increase at a steady rate due to the decay of natural radioactive isotopes such as ^{40}K within the material itself and in the soil around it. When the piece is heated again after its recovery, the amount of light released will be proportional to the number of γ-particles that struck it, and therefore to the time elapsed, since it became buried.

When applied to tiles and bricks of recent manufacture, the luminescence induced by natural radiation should of course only amount to negligible background, and in a sample from Hiroshima and Nagasaki, the lion's share of the signal should come from the intense flash of γ-rays that it was exposed to when the bomb went off. We will consider experimental studies of this kind in Chapter 4.

A.8.4 Mass spectrometry. This method does not measure radiation as such, but it can nevertheless be used to determine the presence and abundance of radionuclides in a sample. As the name suggests, mass spectrometry simply distinguishes atoms—or, in other applications, molecules—according to their mass; it can therefore be used with both stable and unstable nuclides. The method requires that all atoms are converted into single ions, then accelerated in an electric field, and finally captured in a detector. The crucial step for identification is the acceleration: it must overcome inertia, which is proportional to mass; therefore, between two atoms of equal charge but different mass, the lighter one will reach the detector before the heavier one.[17]

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the time, the ball will roll on all the way to the exit; but every so often, it may get stuck at some obstacle along the course instead. To get it rolling again, you have to supply some activation energy by punching the table. In thermoluminescence, the heat provides the punch that frees the electrons trapped in metastable states.

[17] This is the principle of separation in *time of flight* (TOF) mode, which is the easiest to understand; however, mass spectrometry has other modes of operation as well.

Mass spectrometry is very powerful and versatile; nevertheless, it has not fully replaced radiation counting. To understand the respective advantage of each method, consider that radioactive isotopes decay on vastly different time scales (Section A.3.1). Among the fission products of ^{235}U, a short-lived nuclide is ^{131}I, which has a half-life of 8 days, whereas a long-lived one is ^{129}I, which has a half-life of 16 million years. (Both are isotopes of iodine.)

Assume we have a sample that contains 1 ppm (one millionth) of ^{131}I, and the remainder of ^{129}I. Mass spectrometry will simply count the nuclei as they are at any given moment, and will give us the true abundance right away—but the very small fraction of ^{131}I in our sample might get lost in the noise. On the other hand, if we use radiation counting, the far shorter half-life of ^{131}I means that many more of its atoms will decay during the time interval of the measurement—indeed, even at these odds, its signal will be about 700 times higher than that of ^{129}I; and with a small sample, we might entirely miss the ^{129}I. This effect is no mere curiosity; for example, in order to measure uranium isotopes in soil samples, radiation counting would be preferred with the relatively short-lived ^{234}U, whereas mass spectrometry would be more suitable for the longer lived isotopes ^{235}U or ^{236}U.

A.9 Radiation dose

We have seen that ionizing particles can interact in various ways with matter. While these distinctions are often important, it is also useful to have a global measure of the overall dose of radiation received by a target, and in particular by living organisms. Since each of the interactions between radiation and matter involves some transfer of energy, we can use the sum of all the energy transferred to measure the total dose. The unit of measure is the Gray, or Gy for short; 1 Gy is defined as $1\,\mathrm{J}/1\,\mathrm{kg}$.

To understand how much, or rather how little, energy 1 Gy actually amounts to, consider this: 1 J is approximately equal to 0.25 cal, and thus will heat one gram of water by 0.25°C. Accordingly, a kilogram of water that receives a radiation dosage of 1 Gy will thereby be heated by approximately 0.00025°C. With ɣ-radiation, the lethal dose in humans is on the order of 8 Gy; therefore, a lethal dosage of ɣ-radiation will heat up the body by an entirely imperceptible 0.002°C. Thus, the total energy associated with a lethal radiation dose is minuscule; it is the very high energy associated with each of the individual ionizing particles that makes them so fearfully effective.

A.9.1 Dose and Kerma. We just saw that the dose is defined in terms of energy transferred from ionizing particles to a unit of target mass. In this context, one

Table A.1 Relative biological effectiveness (RBE) of different types of ionizing radiation

Radiation type	RBE
α-particles	20
β-particles	1
photons (γ-rays and X-rays)	1
neutrons	5

can make a subtle distinction: the energy thus transferred may remain in that target mass unit, or it may escape it in the form of secondary radiation (see Section A.7.2). The escaping fraction of the energy is included in the *kerma*, which is an acronym for 'kinetic energy released per unit mass', but is excluded from the dose.

How important is this distinction with human bodies? We have relatively large bodies; therefore, much of the energy that will escape one kg-sized portion of our body will end up in the next, and vice versa. Therefore, fruit flies and silkworms probably have more reason to worry about the difference than we do; for the purpose of this book, we can treat the two as approximately equivalent.

A.9.2 Biological effectiveness of different particle types. Qualitatively, all types of radiation induce the same kinds of genetic damage in cells (see below); however, if we use identical doses of each as measured in Gy, then the extent of the damage will vary considerably. To account for this, biological weighting factors have been distilled empirically for each type of radiation from experimental observations (Table A.1). These weighting factors go by various names; we will here adopt *relative biological effectiveness* (RBE). In order to estimate the biological effect of a given physical dose of radiation, one multiplies the physical dose in Gy with the appropriate RBE:

$$\text{biological dose (Sv)} = \text{RBE} \times \text{physical dose (Gy)}$$

Since the Q factors are dimensionless, the unit of the biological dose—the *Sievert*, or Sv for short—is also equal to $1\,\text{J}/1\,\text{kg}$, as is the Gray. Which unit to use depends on the context. It probably goes without saying that the numbers listed in Table A.1 are approximations. With neutrons, there is considerable debate. In Figure 5.1, we use the dose-adjusted RBE described by Sasaki et al. [72], but the neutron RBE value listed here, 5, is a reasonable approximation in the relatively high dose range that matters most in this book.

A.10 Forms or radiation released by fission bombs

While fission bombs may of course be detonated anywhere, we will confine the discussion to air bursts at considerable altitude, as allegedly occurred in Hiroshima and Nagasaki.

A.10.1 Immediate radiation: γ-rays and neutrons. While inside the bomb itself there is a veritable stew of particles (see Section A.5), the β-particles and the fission fragments have low ranges within the bomb and even within air, and they will not contribute to radiation on the ground. In contrast, both γ-rays and neutrons can escape the bomb and strike the ground; it is these two particles that account for the intense yet short-lived burst of immediate radiation from the bomb. Exactly what share of the neutrons will escape the bomb and contribute to radiation on the ground remains uncertain and contentious. For several decades after the event, it was proclaimed that at Hiroshima the biological dose due to neutrons had roughly been on par with that due to γ-irradiation, but later on the neutron dose was revised downward to an almost negligible quantity [79]. This strange story will be examined in a later chapter.

A.10.2 Nuclear fallout. With fission bombs of the size used in Japan, the fireball of the detonation is expected to reach a maximum diameter of approximately 200 m. Since both bombs were set off at an altitude of at least 500 m, the fireball did not touch the ground.[18] Most of the radioactive witches' brew therefore did not come down in Hiroshima itself, but was instead carried upward in and away by the thermal updraft that was caused by the heat released by the bomb itself. However, some fraction *did* reach the ground as local fallout, carried at least in part by the black rain already mentioned in Section 1.2.

A.10.3 Induced radioactivity. Neutrons released by the detonation will strike the ground and, often after first losing most of their energy through a series of collisions, they will be captured by some nuclides on the ground. In many cases, the new nuclides formed by the capture will be radioactive; and since they will tend to have a neutron surplus, they will undergo β^--decay, which is often accompanied by significant γ-radiation. Interest in this *induced radiation* is twofold:

- at least for a short time after the detonation, some very short-lived nuclides may contribute to the radiation dosage received by people on the ground;
- since radionuclides will be induced in proportion to the intensity of the neutron radiation from the blast, the abundance of the longer-lived isotopes

[18] In contrast, the 'Trinity' test explosion in New Mexico is said to have been detonated at low altitude and to have caused intense radioactivity on the ground [128]. xref

can be used to estimate the neutron dosages that would have been received during the blast.

As noted in Section A.4.4, the efficiency of neutron capture varies with both neutron energy and with the precursor nuclide in question; some precursors capture only high-energy neutrons, others only or preferentially low-energy neutrons. Comparing the abundance of nuclides that would have been induced by neutrons of low and high energy, respectively, can give an indication of the neutron energy spectrum; this is of interest in the estimation of radiation dosages. xref

A.11 Biological radiation effects

We have already seen that ionizing radiation converts molecules to radicals (Section A.4.1). An abundant and particularly reactive radical species is $^{\bullet}$OH, which is formed from water. While $^{\bullet}$OH reacts with virtually anything in the cell, including protein molecules and cell membranes, its most significant target is DNA. This is not due to any particular chemical reactivity of DNA, but solely to its special biological function. Other molecules, when damaged, can always be replaced, but DNA cannot—it is passed on from one generation of each cell and each organism to the next, and thus it must be safeguarded from any damage, since even a small chemical change to a stretch of DNA (a gene) can cause a heritable *mutation* with potentially grave consequences.

Living organisms have been exposed to natural radiation throughout evolution, and accordingly they have developed a fairly elaborate machinery for coping with DNA damage by radiation. This machinery continually scans the DNA for damage. If damage is found, the response to it depends on the extent. If the damage is deemed limited, then the cell will attempt to repair it. In many cases this repair will be completely successful and restore the native, intact state of the DNA; the chances for this are good if one of the two DNA strands has remained unaltered and can therefore serve as a template in the repair of the other. On the other hand, if *both* strands of a DNA molecule are severed, the cell may still succeed in repairing the break and restoring an intact DNA molecule, but the all-important nucleotide sequence may have been altered on both strands. Once this happens, the lesion will have become permanent—a mutation has occurred that will now be passed on to all daughter cells.

A $^{\bullet}$OH radical can readily break a single DNA strand, and if the local concentration of such radicals is high enough, then two breaks may occur simultaneously on opposite strands, producing the double strand break situation

described above.[19] In this context, it is interesting to note that, although the underlying chemistry is different and no •OH radicals are involved, sulfur mustard can also produce DNA double strand breaks [100], which can explain the striking similarities of its biological effects to those of ionizing radiation.

While DNA repair may seem like an 'obvious' coping strategy, a more surprising one is *apoptosis*, or programmed cell death. Each cell in the human body that contains DNA[20] will commit *harakiri* when the load of DNA damage, and therefore the chance of harmfully mutated progeny, becomes too great. A key effect observed in apoptotic cells is the destruction of the cell nucleus, which contains the DNA; this can be observed by conventional light microscopy, but also at the molecular level as DNA fragmentation.

Intriguingly, cells in different tissues differ significantly with respect to the level of DNA damage beyond which they will abandon repair and initiate apoptosis instead. This tissue-dependent threshold largely accounts for the observed order of organ damage in radiation. Among major organ systems, the bone marrow is affected first, and with it the regeneration of all types of blood cells; mucous membranes in the intestine are the second most susceptible. And again, since this response to DNA damage is built into the various tissues themselves, it is understandable that DNA-damaging agents other than radiation (such as sulfur mustard, of course) will produce a very similar pattern of organ damage.

Differences in radiosensitivity exist not only between tissues but also within them. In a tissue that actively regenerates, the cells form a continuum of subpopulations, which ranges from rapidly dividing, undifferentiated cells to those that no longer divide but are fully differentiated (Figure A.6). The most rapidly dividing cells are also the most sensitive to radiation; the differentiated cells, which have acquired all tissue-specific traits they need to function as that tissue's 'worker bees', have low sensitivity to radiation.

If the tissue is exposed to a relatively low radiation dose, then only the most sensitive, least differentiated cells may be killed off. The partially differentiated cells will go on maturing and sustain the tissue function a while; this corresponds to the clinical observation of a *latency period*, during which an irradiated patient may appear to be stable or improving. A higher dose will harm some partially differentiated cells also, and therefore shorten this clinical latency pe-

[19] This is the reason why α-particles, which deposit all their energy along a very short distance and therefore produce a high local •OH concentration, have a very high relative biological effectiveness.

[20] Red blood cells and blood platelets don't contain DNA, and thus are exempt. The precursor cells of the red blood cells, however, which reside in the bone marrow, do contain DNA and accordingly are subject to apoptosis.

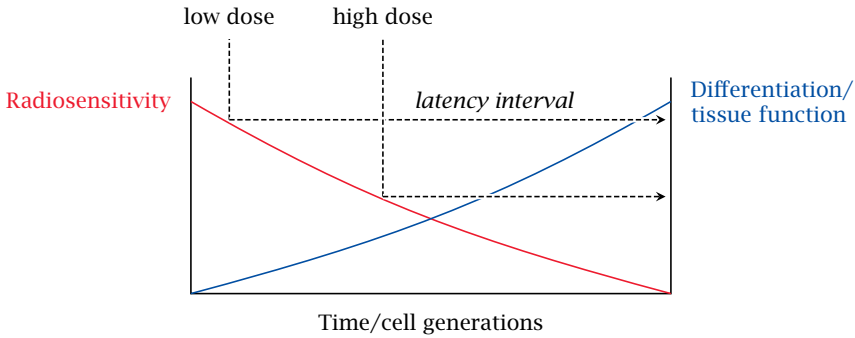

Figure A.6 Radiosensitivity and differentiation of cells in tissues. Within most tissues, there exists a continuum of cells at different stages of differentiation. The least differentiated, most actively regenerating cells are also the most susceptible to radiation. A higher radiation dose thus will deplete more highly differentiated cells, and thus shorten the latency interval after which the lack of fully differentiated, functional cells becomes clinically manifest.

riod. Moreover, it will more likely kill off every last one of the undifferentiated cells, the *stem cells*, from which all differentiated ones originate, and thereby cause irreversible, lethal damage to the tissue. These observations are directly relevant to acute radiation sickness.

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